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MANUAL

OF THE

PHYSICAL DIAGNOSIS

OF

DISEASES OF THE HEART

INCLUDING THE USE OF THE

SPHYGMOGRAPH AND CARDIOGRAPH

BUREAU OF MEDICINE & SURGERY
APR 7 1881

BY

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THIRD EDITION

PHILADELPHIA
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PREFACE

TO

THE THIRD EDITION.

THE favourable reception accorded to former editions has convinced me that my little work supplied a want. It was my aim to give in a concise form the essentials for the diagnosis of Diseases of the Heart according to the most modern teachings of pathology, tested, confirmed, and extended, where possible, by personal investigation. I wished that my book should be a companion to the advanced student in the wards of the hospital, and an *aide-mémoire* to the practitioner in his daily duties. Although the second edition became rapidly exhausted, cardiac pathology has made considerable progress since its publication; it has, therefore, been absolutely necessary to subject the whole to thorough revision. To my regret, nearly a twelvemonth has passed before I have been able to complete this third edition.

The day has come when every educated practitioner should be familiar with the use of the sphygmograph and cardiograph. The difficulties in the use of these instruments, and especially the objection on account of the time which their application occupies, are vanishing, owing to improvement in mechanism; whilst the value of the evidence which they afford is attested by daily experience. As regards the systematic inves-

tigation of disease by such methods, much remains to be done, but, thanks to the labours of Drs. Galabin, Mahomed, Balthazar Foster, and others, as well as to the introduction by Dr. Pond of an instrument which can be employed readily and rapidly, data are rapidly accumulating. I have, therefore, endeavoured in this edition (though in the former I expressly excluded the subject as premature) to describe the methods of employment, and so far as observations warrant, the indications of the sphygmograph and cardiograph in cardiac diagnosis. The illustrations of this portion of the work are for the most part original, from tracings by myself or by my house-physicians and clinical assistants under my own direction. Among those who have rendered me efficient aid I would especially mention Drs. S. D. Clipping-dale and Needham, and Messrs. Major Greenwood, Basil W. Walker, Burry, and Sweeting.

I indulge in a hope that I may be enabled to follow up this little work with another on the *Treatment* of Heart Disease, so arranged that the two volumes shall constitute a systematic work.

SCHEMA.

I.—Symptomatology.

A. Symptoms referred to the Heart. (*a.*) *Pain* referred directly to the Heart-region in cardiac disease is rare. Organic disease may progress without giving rise to Pain. The special pain of Heart Disease is *Angina Pectoris*, characterized by paroxysmal recurrence, great distress, coldness, arrest of respiration. (*b.*) *Palpitation:* A frequent symptom in Heart Disease, but common in dyspepsia and in emotional conditions. (*c.*) *Intermission:* Common in Heart Disease, but may be neurosal. (*d.*) *Irregularity:* Generally of grave import, but may also be neurosal.

B. Symptoms referred to the Circulation. (*a.*) *Pulsation:* Excluding emotional causes, suspect Hypertrophy of Heart, and especially *aortic regurgitation.* (*b.*) *Hæmorrhage:* Common in Heart Disease: Not of dangerous import as in Phthisis: *Note* tendency thereto in *mitral stenosis.* (*c.*) *Cyanosis: Vide Inspection.* (*d.*) *Dropsy:* A late and dangerous symptom.

C. Symptoms referred to the Lungs. *Note* that these symptoms are very frequent. (*a.*) Dyspnœa, aggravated by exertion; periodic or persistent. (*b.*) Cough.

D. Symptoms referred to the Brain. (*a.*) Languor and powerlessness. (*b.*) Vertigo and symptoms of disturbance of cerebral circulation. (*c.*) Epilepsy. (*d.*) Chorea. (*e.*) Apoplexy. (*f.*) Paralysis.

Note.—In cases of cardiac hypertrophy co-existing with renal disease, there may be intra-cranial hæmorrhage, sometimes difficult to distinguish from uræmia. In sudden cerebral attacks in patients with valvular disease suspect Embolism.

E. Symptoms referred to the Alimentary Canal. (*a.*) Dyspepsia, very common. (*b.*) Hæmorrhoids.

F. Symptoms referred to the Throat. (*a.*) Pain referred to the throat may be a variety of *Angina.* (*b.*) Aphonia occasional in pericarditis. (*c.*) Hoarseness.

G. Symptoms referred to the Kidneys. (1.) Renal disease may be induced by the Heart-affection. 2.) Renal disease may induce hypertrophy of the Heart. (3.) Renal and cardiac disease may be the double effect of one cause.

NOTE.—In cases of Cardiac Disease, always examine the urine, and especially record conditions of albuminuria.

II.—ETIOLOGY.

(*a.*) *Rheumatism* is the most frequent cause of valvular Disease of the Heart. *Rheumatic Fever* is in a large number of cases the starting-point, but in other cases the rheumatic symptoms may be very slight and obscure, and sometimes the rheumatic form of endocarditis may occur with no other manifestation of rheumatism.

NOTE.—Examine the condition of the heart in the slight, as well as in the severe forms of Rheumatism.

(*b.*) Scarlet Fever is also a cause of valvular disease and of pericarditis, probably because of the rheumatoid phenomena associated with it. Measles also may be followed by heart-disease. (*c.*) The other most common causes of Heart Disease are muscular overstrain, alcoholism, syphilis, tuberculosis, the puerperal state, poisoning by phosphorus, mal-nutrition, disease contiguous to heart and pericardium, diseases of lung inducing venous engorgement.

NOTE.—A satisfactory examination of any patient, whatever his ailment, cannot be made unless the cardiac conditions are observed and recorded.

III.—PHYSICAL EXAMINATION—INSPECTION.

A. Hue of the surface. (*a.*) Blueness. (1.) *Congenital cyanosis* indicates persistent foramen ovale, or imperfection of inter-ventricular septum, usually combined with obstruction of pulmonary artery. (2.) *Intermittent cyanosis*, common in cardiac dyspnœa.

NOTE.—Chilling of finger-tips with blueness and coldness common in Heart Disease.

(*b.*) Pallor. (1.) When associated with œdema, suspect co-existence of *renal disease*. (2.) With exophthalmos, thyroid enlargement and irritability of Heart, *Graves' Disease*.

(*c*) Tinge of Jaundice. (1.) In passive congestion of liver in later stages of valvular disease. (2.) With *arcus senilis* in *Fatty Degeneration of Heart.*

B. Cardiac Dyspnœa. (1.) Characterized by gasping air-craving, aggravated by exertion—Orthopnœa; indicative of valvular diseases, or cardiac degenerations.

(2.) Decubitus on right side, with expression of anxiety and apprehension. Suspect *Pericarditis.*

C. Œdema: Commences in lower extremities, serous cavities affected last: Usually depends on *valvular disease* (especially mitral, or conjoined mitral disease and tricuspid insufficiency).

NOTE.—Examine urine, and determine the question of co-existence of renal and cardiac disease.

D. Pulsation. (*a.*) In veins of neck indicates *tricuspid insufficiency.* (*b.*) In arteries: Locomotive pulse indicates *aortic regurgitation.* (*c.*) Displacement outwards of visible apex-beat indicates *hypertrophy* or *dilatation of left ventricle.*

NOTE.—Area of visible impulse may be apparently increased at times in nervous palpitation.

(*d.*) Retraction of intercostal spaces coincidently with systole indicates *pericardial adhesions.*

(*e.*) Pulsation of the liver indicates *tricuspid regurgitation.*

IV.—PHYSICAL EXAMINATION—PALPATION.

A. Pulse. (*a.*) Rigid arteries, tortuous, with roughnesses felt in their walls, associated with hard pulse, indicate *Atheroma.*

(*b.*) Slowness of pulse, with very weak impulse, may indicate *Fatty Degeneration.*

(*c.*) Sudden variation of pulse with great acceleration on movement may occur in *Pericarditis.*

(*d.*) Jerking, collapsing pulse in *aortic regurgitation.*

(*e.*) Feebleness of pulse, and (*f.*) Irregularity and intermission, increased by effort, in *mitral disease* and *cardiac degeneration.*

B. Apex-beat. Displaced. (1.) Upwards by *pericardial effusion.* (2.) Downwards and outwards in *hypertrophy* and *dilatation* of heart.

C. Forcible pulsation felt under false ribs to left of ensiform cartilage indicates *hypertrophy of right ventricle.*

D. Visible pulsation of right auricle indicates *hypertrophy and dilatation of right chambers.*

E. Visible pulsation of left auricle indicates *mitral stenosis.*

NOTE that this pulsation may be rendered evident by vibrating levers.

F. Special Cardiac Tactile Phenomena. (*a.*) Friction-fremitus denoting *Pericarditis.* (*b.*) Thrill. (1.) Occurring with systole over aortic valves denotes *aortic stenosis* or *aneurism;* over pulmonary valves, *pulmonary stenosis;* at apex, *mitral regurgitation.* (2.) Occurring with diastole at base, denotes *aortic regurgitation.* (3.) Occurring at apex just before, and terminated by impulse, indicates *mitral stenosis.*

NOTE that decided presystolic thrill at apex is pathognomonic of mitral stenosis, and may occur without murmur.

V.—PHYSICAL EXAMINATION—PERCUSSION.

A. Præcordial area over-resonant or tympanitic—in emphysema of lung, and in the very rare cases of pneumo-pericardium.

B. Præcordial dulness extended. (*a.*) Upwards; outline of dull area being triangular or pyriform with apex at or above third costo-sternal articulation. There is probably *Effusion into pericardium.* (*b.*) Laterally. The heart is enlarged by hypertrophy or dilatation.

I. Lateral dulness extends *left* of normal area. (1.) Area of dulness triangular, apex pointed, *Hypertrophy of Left Ventricle.* (2.) Area of dulness rhomboidal, apex rounded, *Dilatation of Left Ventricle.*

NOTE.—Compare position of apex and outline of left ventricle with evidences of force of apex-beat. If left side enlargement with forcible heaving impulse and long first sound, *Hypertrophy.* If enlargement with feeble impulse and short first sound, *Dilatation.*

II. Lateral dulness extends *right* of normal area. There are *Hypertrophy and Dilatation of Right Auricle and Ventricle.*

NOTE epigastric impulse and signs of tricuspid regurgitation, if present.

VI.—Physical Examination—Auscultation.

Section I.—The normal heart-sounds are modified in degree.

A. At the *base* of the heart:—

(*a.*) The aortic second sound is intensified. There is *Hypertrophy of the Left Ventricle.*

(*b.*) The pulmonary second sound is intensified. There is heightened tension in the pulmonic circulation.

Note.—A strong pulmonary, associated with a weak aortic second sound, is presumptive evidence of *mitral obstruction or insufficiency.*

B. At the *apex* of the heart:—

(*a.*) The first sound is increased in duration. There is *Hypertrophy of Left Ventricle.*

Note.—When there are signs of Hypertrophy of Left Ventricle without other cardiac signs to account for it, suspect *Chronic Renal Disease.*

(*b.*) The first sound is short, resembling the second sound. There is *feebleness, dilatation or degeneration of left ventricle.*

Note.—Reduplication of heart-sounds occasionally occurs in health; when associated with fever or with grave debility, indicates *myocarditis or cardiac degeneration;* if reduplication of *second* sound, suspect *mitral stenosis.*

Section II. Abnormal sounds are heard over the heart-region.

A. Generally over the præcordial area.

(*a.*) Friction sound indicates *Acute Pericarditis*—or *Pericardial roughening,* the result of a remote pericarditis.

B. Localized in definite relation with the situations of the valves.

(*a.*) First-sound murmur over site of aortic valves (anæmia excluded) indicates *aortic obstruction.*

Note.—The murmur may be localized or propagated in the course of the great arteries.

(*b.*) Second-sound murmur over site of aortic valves indicates *aortic regurgitation.*

Note.—This murmur may be localized, or propagated downwards in the line of the sternum.

(*c.*) Double (first and second sound) murmur (*a* and *b*) denotes combined *aortic obstruction and insufficiency.*

(*d.*) First-sound murmur over site of valves of pulmonary artery denotes (anæmia and pressure upon trunk of pulmonary artery excluded) *obstruction of pulmonary artery.*

NOTE.—Pulmonary obstruction is nearly always congenital.

(*e.*) First-sound murmur at base of ensiform cartilage indicates ***tricuspid regurgitation.***

NOTE.—This is most commonly found as secondary result of mitral disease.

(*f.*) Before first-sound murmur at base of ensiform cartilage denotes ***tricuspid stenosis.***

(*g.*) First-sound murmur heard over situation of right ventricle may be due to *myocarditis* or *anæmia with cardiac debility.*

NOTE.—These murmurs are not persistent.

(*h.*) First-sound murmur localized in mitral area denotes ***mitral regurgitation.***

(*i.*) Before-first-sound murmur localized at apex, or just internal thereto, denotes *mitral stenosis—i.e., obstruction.*

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PART I.

THE ORDINARY METHODS OF PHYSICAL DIAGNOSIS.

LECTURE I.

INTRODUCTORY.

Symptomatology—Importance of subjective symptoms in regard to treatment: of objective in diagnosis — Pain — Angina pectoris — Cardiac symptoms — Disturbances of circulation—Pulmonary symptoms—Cerebral phenomena—Embolism—Gastric symptoms—Signs referred to the throat—Renal symptoms—Etiology—Rheumatism—Other causes of heart disease.

A FEW words on the threshold of the subject—advice pertinent to investigation of disease in any form, but yet I think rather particularly so to the class of diseases which we shall presently examine. When a suffering patient comes to you for examination, do not permit a diversity of purpose of intent to exist between you mind and his. His chief object is to get relief from his aches and pains; yours, perhaps, is to find out—by a process similar to that employed by botanists to discover the name of a newly found flower—the name of the disease which he suffers from. Remember that your duty is not chiefly to find out what disease he is the subject of, but what his sufferings are, what his deviations from sound health. The plane of unhealt

is divided into definite squares, which are called Diseases, and the nomenclature of these is very important, because the mention of one of them displays before the medical mind a large picture, and a moving panorama of phenomena; but your sole object is not to find out into what particular square your patient's aggregate of symptoms will fit. You must investigate his ailments as well as his diseases if you would do him good. I am very far from wishing to be a censor, but I do think that in these days of great and increasing precision in physical diagnosis, there is a real danger of students thinking too lightly of the uttered complaints of patients. It has been said that one objective sign is worth a dozen subjective symptoms, and this, so far as regards diagnosis, has a large amount of truth; but we must recollect that perturbations, which greatly distress our patient, may exist independently of his chief disease, and it is the patient, not the disease, that we have to treat. Let me instance a hypothetical case. A. B. comes to you, and, under fire of cross-examination discloses that he has had rheumatic fever some years ago. You examine his heart and find something wrong with the mitral valve. You dose him, probably help his heart over its presumed difficulties with digitalis, and order the inevitable belladonna plaster to be applied. But your patient at his next visit is not a whit better. It was not his heart which troubled him—that was neither better nor worse than it had been for many years past, and was likely to be for many years to come; but he suffered from a *trigeminal neuralgia*, and would have showered blessings upon you had you relieved him.

The moral I would point, then, is this: Always

commence your interrogatory of a patient by asking what he complains of, what are his troubles and distresses, and if you record his case, write on the first line, *complaint*, and give his expressions, as nearly as possible, with the necessary abbreviations, in his own words.

Now we come more closely to our subject. The comprehensive view of what disturbs the equanimity of a patient, with a view to the alleviation of his troubles is one thing, the particular determination of the physical significance of his ailments, and the precise diagnosis of his diseases is another. For this latter purpose, the details of pains and aches, though all due weight must be given to them, are very deceptive. How very common it is for a patient to fancy that he has disease of the heart because he experiences discomfort or pain in the heart-region, and to insist on being "sounded," so that his doubts may be set at rest. How rarely do we find that he really has organic heart disease? Oftentimes the patient goes away apparently with a look of disappointment that his fears are not confirmed, and with a doubt probably of our skill, because his aches are so precise.

In looking over the notes of a hundred cases of the coarse and decided forms of disease of the heart which have been under my own immediate care, I find that in just half the number, there was no complaint whatever of any pain referred to any part of the chest. Seventeen referred the pain generally to the front of the chest, fifteen to the back of the chest, and especially between the scapulæ. Twelve referred the pain to the epigastrium; eleven suffered pain on the left side of the chest; whilst two referred their sufferings to the right side. Those who localised their suffer-

ings to the exact area of the heart were but eight; and of these, two complained of it only on exertion; one referred it to the base of the heart; one described it as a sense of extreme soreness at the apex; and in one, it partook of the character of neuralgia of the left breast. So far as the evidence derived from the above cases—which are, I believe, fair specimens of those presenting themselves at the public or the private consulting room—teaches only eight per cent., or less than one in a dozen, complain of pain directly referred to the situation of the organ which is diseased. This, though strange to the non-medical mind, can scarcely be surprising when we consider that the heart possesses very little common sensation, and that its structure can be punctured, torn, or lacerated, without the direct infliction of pain. When we consider the vast number of patients who present themselves with symptoms referred to the region of the heart, but really caused by dyspepsia or pleurodynia, and then take into account the very small proportion of real undoubted heart cases presenting signs of local pain, we must estimate at a very low figure the value, in a diagnostic point of view, of *pain* in relation to heart disease.

Looking on diseases of the heart in general from the point of view of symptomatology, we find that their origin and progress are oftentimes very insidious and obscure. Even the most pronounced and dangerous forms of the disease may, in some cases, go through their stages without betraying themselves by marked symptoms of distress. This fact was strongly impressed upon my mind by a case which was under my care at the North-Eastern Hospital for Children. A little girl, who had been treated pre-

viously for a valvular affection, was brought to me because the mother thought she seemed a little languid, though the child complained of no distress. On examination, I found that there was effusion into the pericardium. She was admitted, and went through all the stages of pericarditis with extreme distension of the pericardium, exhibiting one of the most pronounced instances of pericardial friction that I have ever heard and an accompanying endocardial change, and yet throughout her whole illness the child complained neither of pain nor distress, and it was impossible to keep her in bed.

There is one form of pain in heart disease, however, which is of terrible significance—I mean Angina Pectoris. It has the character of a grip; it shoots down the left arm and occasionally involves both arms, or radiates to the back.* This is the pain, *sui generis*, of cardiac origin, and if you have once seen a pronounced example of it, you will never forget it. The patient suddenly sits up in his bed, and with a cry of horror indicates his sense of pain at the præcordium. It has great intensity, but is of a cold and

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* "The pain of angina is distinctly located in or about the midsternum, whence it radiates. Eulenberg says this is due to the connection between the superior cardiac nerve and the anterior branches of the four upper cervical nerves; while the middle and inferior cardiac nerves are connected with the four lower cervical nerves, uniting in the brachial plexus and first dorsal nerve. The pain usually runs out at the peripheral endings of the ulnar nerve, especially the little finger. It is almost invariably found on the left side only. In a case where it was on the right side, the pulmonary artery was the seat of disease."—"Path. Soc. Trans.," 1878. "The Diseases of the Heart," Dr. Milner Fothergill. Second edition, London, 1879, p. 285.

sickening character: the chest is fixed, the breathing not quickened, and your hand, placed over the epigastrium, finds that the heart's action is slow and laboured. The face wears a look of horror, the hue is pale or slightly leaden, and a cold sweat breaks out upon the forehead. Worse than the pain is the feeling of fearful sinking and depression; the poor patient gasps, "I shall die!" and, sometimes, as in a case which it was once my lot to witness, his short, but concentrated sufferings in a few minutes end in death. From such a typical case of high diagnostic and prognostic import, there are many gradations of intensity, until, in some cases, it is difficult to differentiate the symptoms from those of dyspepsia or hysteria. In some cases the pain is absent though the other symptoms are present; such is the "angina sine dolore" of Gairdner. The points to be remembered are:—(1) The attacks are paroxysmal with long or short intervals. (2) There is always a sense of coldness experienced, and frequently a cold sweat. (3) The heart's action is *not* increased, and (4) The chest is fixed, the breathing slow.

Few questions in the domain of medicine have presented greater difficulties than that of the nature and pathological significance of angina pectoris. Latham said of it: "We are sure of what it is as an assemblage of symptoms. We are *not* sure of what it is as a disease." In the fatal cases, the lesions which have been discovered have been various, and in some no morbid appearances have been demonstrated at all. The most commonly discovered conditions have been (*a*) degeneration and calcification of the coronary arteries; (*b*) atheromatous disease of the aorta; (*c*) disease of the aortic valves; (*d*) fatty degeneration of the muscle of the heart. The affection is rare, though by no means unexampled, be foremiddle age, and is much more common in the male than in the female sex. It has proved fatal even in a first attack; Dr. Arnold, of

Rugby, died within three hours of the onset of an attack of angina pectoris. In the case of a young man, aged twenty-three (recorded by Dr. John Wilson, *Edinburgh Medical Journal*, September, 1874), who died the day after he had manifested heart-pang, no disease was found at the post-mortem examination. The heart was flaccid, the cavities empty, and the valves all healthy. The young man had been in perfect health until a sudden exposure to cold led up to the fatal attack. It is obvious, therefore, that we must not look alone to the post-mortem table for a solution of the difficulties which beset the pathology of angina pectoris. Fortunately, clinical investigation has succeeded in a large measure in dissipating those difficulties. Dr. Lauder Brunton demonstrated, in 1866, by means of the sphygmograph, that the *arterial tension during an attack of angina pectoris is increased*. Dr. Brunton, knowing that the effect of nitrite of amyl was to relax the arterioles and thus to relieve the tension, administered this drug by inhalation during the paroxysm of angina, with complete success; the symptoms were immediately alleviated. Dr. L. Brunton's observations have been conclusively confirmed. More recently, Dr. Murrell has introduced for the treatment of angina pectoris another agent, which, like amyl nitrite, has the power of relaxing the arterioles—nitro-glycerine. The use of this drug, which is administered by the stomach, is also attended by most obvious relief of the symptoms. It may, therefore, be taken as proved, I consider, that angina pectoris is essentially a condition in which the arterioles are contracted, and in which there is high pressure in the arterial system, and that when the arterioles are made to dilate, and the intra-vascular pressure is relieved, the symptoms abate. You should remember that angina pectoris is by no means a common affection. Dr. Hayden, in his large experience, states that he has met with only six genuine examples ("Diseases of Heart and Aorta," p. 1043). It follows, therefore, that you must be careful, in any case which appears to you to present the characteristic symptoms, to discriminate between true angina and the many forms of pain which are sometimes called *false* angina, and which may be neuralgia, intercostal rheumatism, the consequences of dyspepsia, &c. You will make the diagnosis by observing whether the symptoms correspond

with those I have before described, and especially also by ascertaining, if you have an opportunity of witnessing the phenomena of an attack, whether the arterial tension during the paroxysm is increased. On this point you should obtain the evidence of the sphygmograph if possible. Having recognized your case as one of true angina, supposing that you have opportunities of further investigation, you will find it, in my opinion, most practically useful to place it under one of two categories. *A*, in which the arterial tension is not increased in the intervals of attacks. *B*, in which there is persistently high arterial tension. The cases (*A* in which normal tension is moderate or low, constitute the minority, and for the most part comprise the exceptional cases occurring in the earlier periods of life. In such there is an acute supervention of high pressure, the arterioles become contracted (perhaps in obedience to a direct external cause, such as exposure), the left ventricle struggles against the difficulty so imposed, and becomes overdistended. As I have said, such acute angina may be fatal. On the other hand, the paroxysm being relieved, the patient may go an unlimited time without recurrence. In such cases there may be no antecedent cardiac disease; it is not common to meet with angina in the valvular diseases consequent on rheumatic endocarditis. I have seen, however, a single severe attack in a lady aged thirty-one, who presented the marked signs of mitral regurgitation; in this case, however, the sphygmograph demonstrated high tension, a condition quite different to that usually obtaining in such pronounced mitral regurgitation. In *résumé*, therefore, in Class *A*, the angina attack is a neurosis, the essential of which is the acute supervention of a contraction of the arterioles, with rise of blood pressure throughout the arterial system, and distension of the left ventricle from accumulation of blood therein. In Class *B*, which includes the majority of cases of angina pectoris, the *arterial tension is persistently high*, though it is still higher during the paroxysms. In these there is a special cause for the recurrence of attacks; such cause is disease of the coats of the aorta (aortitis deformans) or disease of the systemic arteries (arteritis) and especially of the coronary arteries. In such cases you will find the arterial pulse hard and incompressible, and there will be signs of hypertrophy of the left ventricle, and probably valvular disease.

which is usually *aortic*. When from any cause the already high arterial tension is increased, and the left ventricle is overtaxed in its struggle to propel the blood, angina may ensue. Undue exertion or effort are frequent exciting causes of an attack. In some cases, however, paroxysms occur without obvious disposing causes, and in such it seems highly probable that there is a direct interference with the nervous mechanism. Lancereaux has described a case which manifested attacks of angina pectoris wherein, at the autopsy, there was found aortitis, with a direct involvement, by the disease of some of the fibrils of the aortic and cardiac plexuses (Cf. Lancereaux, "Anatomie Pathologique," Paris, 1871, p. 257, and *Gazette Médicale*, 1867, p. 432). Seeing the common association of the cases of angina which fall under this section, with disease of the arterial coats, it seems very reasonable to link the nerve-phenomena with an affection of the sympathetic filaments which are in such close relation to the coats of the vessel. If we accept this view, the calcification of the coronary arteries—heretofore supposed to induce the symptoms through the degeneration of the heart muscle, which, by impairing the blood-supply, it occasions—is the cause of the pain rather by the direct implication of the cardiac plexus. It is quite true that cardiac degeneration follows coronary obstruction; but this is a cause, not of the pain, but rather of the fatal issue. In a case recorded by Romberg, it was proved that not only the cardiac nerves were involved, but also the vagus. In his attacks, the patient, a man aged thirty-six, after a prodroma of apprehension and terror, felt his heart stand still—there was an intermission of from five to six beats—at the same time he experienced violent pain on both sides of the chest, extending to the neck and head. Here was a complex form of angina, the peculiarity of which was a long arrest of the heart's action. At the post-mortem examination the great cardiac nerve was found very distinctly diseased, and the left vagus as well as the phrenic were involved in diseased glands. The vagus is the inhibitory nerve of the heart, and there could be no doubt that an irritation of its inhibitory fibres produced the long intermissions of the heart's action witnessed in the case. The high tension observed in these cases is not necessarily associated with hypertrophy of the left ventricle. In the case of Dr. Arnold,

the walls of the left ventricle were much thinner and softer than natural. The great John Hunter died after attacks of angina pectoris, and the heart in his case was found to be very small and its tissue pale. Dr. W. T. Gairdner says: "Post-mortem examinations have generally shown that the heart is found flaccid rather than rigidly contracted, and the lesions found in the muscular substance of the heart itself are usually such as would confirm the idea of decidedly and permanently weakened energy, rather than a disposition to abnormal contraction." The high tension, then, is due to vascular rather than to cardiac causes; the left ventricle labours against the obstacle imposed by the firm contraction of the arterioles. In the large class of cases of angina pectoris associated with atheromatous disease of the great arteries, I consider that a strict analogy is established with those cases of epilepsy which are found to depend on local irritation of the nerve centres. In the former, owing to irritation of the nerve fibrils involved in the wall of the diseased artery, there are periodic explosions expressed by the tight contraction of the arterioles and the consecutive heart-struggles, whilst in the latter the expression of the maximum of central irritation is an epileptic fit. In any case manifesting angina pectoris, I counsel you strongly to obtain, by means of the sphygmograph, evidence of the condition of the vessels. I may mention a case in point. I saw, in conjunction with my friend Dr. John Brunton, an old lady of sixty-nine, who suffered from severe pain, referred to the epigastrium and abdomen. This pain might very easily be referred to visceral congestion, but it was to a considerable extent paroxysmal, and there was a marked look of anxiety upon the face with tendency to cold perspiration. The heart sounds betrayed nothing abnormal, but a tracing of the radial gave very important evidence. Not only was there extremely high tension, but the trace bore strongly the characters of aortic obstruction (see tracing of Aortic Obstruction in Part II.). I had no hesitation in concluding that the vessels were extensively diseased, and it seemed to me very probable that the great suffering referred to the epigastric and umbilical regions might be ascribed to disease of the coats of the abdominal aorta. Nitro-glycerine gave relief, and the subsequent history of the case confirmed the diagnosis. Dr. Brunton wrote me: "The old lady has gone on tolerably well

till two days ago, when failure of pulse came on, only one decent beat in ten, but over the heart the sounds were quite normal in strength, rhythm, and note. . . . You will find the case a most interesting witness of the power of the sphygmograph." It is very probable that some of the recognized forms of abdominal pain are but varieties of angina. Whilst ascribing, however, to direct disease of the arterial tissues the causation of a large proportion of those cases of angina which are accompanied by persistently high vascular tension, I do not mean to assert that in some instances the attack may not be initiated by visceral causes. Bergson has narrated a case of angina, in which there was enlargement of the liver, the attacks ceasing after treatment directed to the hepatic disease. (On this as well as many points of interest as regards angina pectoris, see Dr. Milner Fothergill, "The Heart and its Diseases." Second edition, chap. xi. London: Lewis.)

You have seen, then, that a large number of heart cases occur without the manifestation of any pain—that the special pain of heart disease, though of great diagnostic importance, is of comparatively rare occurrence. You may be led to inquire what are the other symptoms that lead up to the suspicion of heart disease; and although we cannot enter at all deeply into symptomatology, we may endeavour to give a brief answer to the question.

In this review of symptoms, in order to give a rough idea of the relative frequency, I shall place between brackets the number of instances in which each has been observed in the hundred cases which I have taken as typical. The first class of symptoms (A), excluding pain which we have already considered, includes palpitation and disturbances of the muscular structure of the heart. *Palpitation* is far from being a pathognomonic sign of heart disease. Its first cause resides often in the nervous system. It is not by any means a sign of heightened functional

activity, but is rather the "spurt" of overtaxed and wearied muscle. Though occurring very often in conditions in which the heart is not structurally diseased, palpitation is a frequent sign and source of trouble in organic heart affections (28). It is specially called forth on exertion, often of the slightest degree, and it occasions much distress; in some cases, especially where the aortic valves cannot close perfectly, the patients complain that the heart beats like a hammer. Another sign of imperfect action of the muscle of the heart is *Intermission*. After a number of pulsations at regular intervals, the heart waits over the whole period necessary for a contraction, and then resumes, to wait again after another interval. Intermittency, like palpitation, may be no sign of structural heart disease; it is in many cases due to an incoördination of nervous actions, and is to be ascribed, not to cardiac, but to cerebro-spinal causes. When it does occur, however, in ascertained organic disease, it is of serious import. It often means that the contraction of the auricles is at certain times so imperfect that they do not fill the ventricles. The necessary stimulus to the ventricular contraction is a sufficient repletion of the cavities. Hence the ventricles wait until the auricles supply them with enough blood. A third trouble of heart-muscle is *Irregularity*. This, also, is due to a want of action, in accord, of the layers of muscular fibre of which the heart consists. The heart does not wait for a whole beat as in intermission, but alters its rhythm irregularly. Sometimes the left and right sides do not contract, or do not complete their contraction, at one and the same moment; then the action may be *reduplicate*. Sometimes there is so much

disturbance that the periods of action and rest cannot be discriminated; the beats are *tumultuous*. Irregularity may, like the other disturbances of heart-muscle, exist independently of organic disease; but when it reaches a high degree, it is one of the strongest evidences of such disease. Patients sometimes describe the irregular action as like the fluttering of birds. One complained to me of a fluttering in his heart "like two pigeons." Such signs are of dangerous import.

The symptoms, however, may be referred, not to the heart itself, but to the next portion of the system of blood-distribution—to the arteries. Patients complain of (B) *Pulsation*. This, we shall see, is very pronounced in the general arterial system when the aortic valves are incompetent to close. It was complained of by my patients as a symptom of distress (3) in the ear whilst lying on the pillow, in the right temple, in the occipital region. Pulsation is, of course, a characteristic sign of aneurism, but this condition we do not intend to discuss. Another arterial symptom in connection with heart disease, but rare and of no considerable diagnostic importance, is *Flushing*. A very important consequence is (C) *Hæmorrhage* (22). This may occur from the lungs (12), when it must be remembered that it has not the dangerous significance of the hæmoptysis of pulmonary phthisis. Considerable quantities of pure blood can be expectorated with the result of relieving the venous engorgement of the lung which is the result of some forms o heart disease. Of worse omen, in my opinion, is the frequent voiding of blood-stained sputa; this occurs usually when the right side of the heart is dilated, and is one of the late consequences of valvular disease.

Bleeding from the nose (5) is far from uncommon in heart disease; hæmorrhage may occur also from the stomach (3), or from the uterus (metrorrhagia) (2). In all these cases the hæmorrhage may occur from the direct rupture of capillaries by the shock of an unduly contracting left ventricle, but much more commonly it occurs from superinduced *venous* congestion. Occasionally the hæmorrhage is not manifest outwardly, but occurs in the interior of organs.

Other signs are referable to plethora of the venous system. The veins may sometimes be seen to be obviously distended, and in some cases, as we shall describe, the large veins pulsate. Patients with certain malformations of the heart exhibit a blueness of surface (D), *Cyanosis*, from the distribution of venous blood by the arterial channels or from venous congestion. A like blueness obtains intermittingly, on account of venous congestion, in the paroxysms of dyspnœa from which patients with heart disease occasionally suffer. A consequence of habitual venous plethora may be (E) *Dropsy*, of which it is well known that cardiac disease is one of the great inducing causes.

Next in order to the obvious disturbances of the heart itself and the channels of blood-distribution, we come to consider the symptoms of disturbance of the functions of the *lungs* in heart diseases. These symptoms are far more frequent than those referred to the heart itself. Nearly half the cases (45) complain of difficulty of breathing. Some (8) are obliged to sit upright (orthopnœa) in order to breathe. A very large proportion of patients with heart disease suffer from cough (45).

A great characteristic of the dyspnœa of heart disease is, that it is produced or aggravated by slight

exertion. The heart may fairly accommodate itself to conditions of rest, but let exertion call upon it for increased action, and it manifests its distress by the imperfect pulmonary circulation, and the consequent dyspnœa. Or the dyspnœa may be periodic, and not induced by voluntary effort. Such attacks are called cardiac asthma. The induced conditions may, however, be not temporary, but chronic. Persistently defective heart's action induces persistently defective pulmonary circulation. The blood tends to stagnate in the lungs. Chronic bronchitis, and, subsequently, emphysema follow, and the trouble may be augmented by œdema of the lungs.

Next we will proceed to notice the *Cerebral* troubles which occur in heart disease. These are very common. The patients complain of languor and extreme weakness (25). Often the muscular weakness is referred especially to the arms; the patients cannot lift weights as they have been accustomed, and the muscles feel powerless. They suffer attacks of giddiness (vertigo) (8), or are subject to faintings (syncope) (6). There may be an undefined nervousness (7), with dread of a fit or some calamity, and lowness of spirits. Headache (5) is sometimes met with, but is not one of the commonest symptoms. Trembling of muscles and the irregular jactitations of chorea (3) are very important to notice. There may be fits epileptiform (2), or epileptic (2), or various forms of paralysis may be found. Lastly, there may be impaired memory and intellectual disturbance of various kinds. In all cases of heart diseases which present cerebral symptoms, the fundus of the eye should be examined by the ophthalmoscope.

The cerebral phenomena observed in heart disease

may, for the most part, be divided into three classes. The first embraces those due to the *chronic* disturbance of balance between the arterial and venous systems which is the result of imperfection of the driving power in the great engine of the circulation. The brain may suffer from deficient supply of arterial blood, or from excess of venous blood, or from these causes variously combined. Arising from these conditions there may be increase of the fluids effused within the intra-cranial cavities, and degenerations of brain tissue, owing to the impaired nutrition.

The second class of cerebral phenomena occurring in heart disease includes those due to intra-cranial hæmorrhage. You must remember that apoplexy is to be feared in cases of hypertrophy of the left ventricle of the heart, the strong muscular contraction distending the arterioles and capillaries to the point of rupture. It is much more to be feared, however, when there are heart hypertrophy and kidney disease combined. In such cases there is not only excess of driving power, but the arterioles have suffered change—they have become brittle and prone to rupture. Out of twenty-two cases of apoplexy, Kirkes found thirteen accompanied by hypertrophy of left ventricle, and fourteen accompanied by renal disease; and Eulenberg, in six cases of apoplexy, found five with contracted kidney and heart hypertrophy.

The third class includes the interesting phenomena now known to be due to the sudden blocking of a cerebral artery by a morsel of coagulum detached from a diseased portion of endocardium, and swept onwards in the blood current until it happens to be arrested in an arterial channel which it is too large to pass through.

The straightest course which such a plug can pursue is from aorta to middle cerebral artery of the left or the right side, and the symptoms produced are hemiplegia, with coma, or aphasia. Sometimes, however, the effects are more chronic, the plugging of the cerebral vessel, and hence the cutting off of nutrient supply, inducing softening of that portion of the brain supplied by the vessel.

Instead of a large plug of this sort, we have reason to believe that small ones which are arrested in the cerebral arterioles occasionally occur, and may account for symptoms for which no cause has been discovered. Thus in chorea, which has a notable connection with disease of the heart, it has been suggested by Dr. Hughlings Jackson that there are embolisms of the arterioles of the corpus striatum and the adjoining convolutions.

Emboli may be carried in the blood current to other parts than the brain; the spleen, liver, and kidney can be thus affected. Capillary embolism of the kidney is probably by no means uncommon. It is suggestive that in two or three cases I have noted, in which chorea occurred in rheumatic endocarditis, frequent micturition was a symptom complained of. Or from the right cavities plugs may be detached and carried into the pulmonary artery, plugging some of its branches in the lung. From this result the appearances which used to be called "pulmonary apoplexy."

We turn now to another set of symptoms in heart affections, those of the *stomach*. Pain referable thereto we have already noticed. A large proportion of patients with heart disease complain of some of the symptoms of indigestion—gastric catarrh is common;

nausea, vomiting, pyrosis, and flatulence are frequent, and occur in a circle, the heart trouble occasioning them, and the symptoms reacting to cause palpitation and heart distress. The other abdominal viscera also partake of the venous plethora induced in heart disease. Hæmorrhoids are frequently met with.

Another set of symptoms in diseases of the heart comprises those referred to the *throat*. This subject presents, in my opinion, a wide and very promising field for observation. Pain beginning at the throat is referred to by some as a very dangerous sign in these affections. There are many instance on record in which a patient has grasped at his throat, evincing signs of acute pain, and has shortly afterwards expired. The sudden throat pains in heart disease are, I believe, for the most part, varieties of angina, and may be discriminated by the rules I have given. Other forms of pain of less laryngeal character and of less intensity are, however, met with, and these are usually accompanied by flatulence and dyspepsia. The "rising in the throat," unaccompanied by pain, is usually either dyspeptic or hysteric. Next to the throat-angina, the symptoms of greatest interest and importance are loss of voice (1) and hoarseness (4). The case of aphonia which I have noted, occurred during the progress of pericarditis—to what was it due? A case which suggests an answer to this question is given by Dr. Morell Mackenzie in his book on "Hoarseness and Loss of Voice." In this instance pericardial effusion was accompanied by aphonia due to paralysis of the abductors of the vocal cords. After the cessation of the pericarditis, the mobility of the affected laryngeal muscles returned. The pathological process whereby such paralysis is brought

about is yet undiscovered. We find that hoarseness occurs as a symptom of aortic aneurism, the disposing cause in such case being pressure of the aneurismal sac on the left recurrent nerve, inducing paralysis of certain of the laryngeal muscles. I have not found, as far as my own experience goes, that hoarseness is prone to occur in aortic valvular diseases. The cardiac conditions accompanying hoarseness I have found to be disease of the mitral valve, with which broncho-pneumonia, or some other form of pulmonary mischief, co-exists. Unilateral paralysis of the intrinsic muscles of the larynx is frequently met with in local disease of the pulmonary texture.

Disease of the *kidneys* may stand as to disease of the heart in a threefold relation. The latter may be cause, consequence, or concomitant. The renal disease may be (*a*) directly caused by the heart-imperfection. The kidneys, like the other viscera, suffer venous engorgement, and, if this be long continued, a low form of inflammation, attended with increased formation of fibrous tissue, may occur in them. In the earlier stages such a state of things is indicated by albuminuria, in the latter by the detection of kidney tube-casts in the urine by the microscope. But (*b*) the heart disease may be primarily caused by the renal disease. When, owing to structural disease, the kidneys are unable to excrete from the blood the urinary solids, the natural consequence is the retention in the circulation of effete material. It is supposed that this material perfunctorily retained, so irritates the arterioles, or the vaso-motor centre which governs them, as to cause them to contract. Such contraction (it seems to me that the term "spasm," which has been used in respect of this

effect, is misapplied) being long kept up, the result is the same as occurs in overtaxed muscle-tissue generally—viz., hypertrophy; so there is induced a peripheral obstacle to the onward current of blood in the arteries. The heart struggles against such an obstacle, and its efforts produce hypertrophy of the left ventricle. Thus, hypertrophy of the left ventricle may even be induced subsequently to renal disease in children. Or it may be (*c*) that the heart disease and the kidney disease are both effects of one cause. The form of kidney disease which usually accompanies cardiac hypertrophy is what is called "contracted kidney;" this is for the most part associated with some gouty affection. We know that in gouty disease the blood is impure, and it is supposed that such condition gives rise to a disease of arteries and capillaries generally throughout the system leading to thickening of their walls, but not necessarily nor wholly of the muscular part thereof. The heart hypertrophies because it struggles against the obstacle, not of arterioles actively contracting, but of arterioles which have undergone degeneration, whereby their walls have become thickened and inelastic. The capillary circulation is thus rendered slower, and the heart muscle becomes stronger to overcome the impediment so produced. The kidney is diseased because its vessels are involved in the general disease. The rules you can deduce from a consideration of the whole subject are: 1. In cases of cardiac disease carefully examine the condition of the urine. 2. When you find renal and cardiac disease co-existing, weigh carefully the facts of the previous history, and endeavour to find out which pathological condition preceded the other.

I turn now to another branch of the subject. I have said that the symptomatology of heart disease is often obscure. We find that its etiology is often obscure likewise.

There are two errors, in my opinion, into which many are prone to fall. The first is that valvular disease of the heart is rarely found except as a consequence of rheumatic fever; the second that there is danger of heart-complications in the severer forms of rheumatism only. Let us turn to the records of actual cases. Taking seventy-seven of the hundred cases before cited, in which the early histories are sufficiently precise, I find that thirty-four occurred in those who had suffered one or more attacks of undoubted rheumatic fever; but in thirteen there had been rheumatic pains only, not sufficient to keep the patients to their homes; and in fifteen there was no history of any rheumatic affection whatever, and only, if any symptoms at all, those of a lightly-regarded indigestion. Rheumatic gout had been suffered by two patients, scarlet fever by three, and typhoid, or "low" fever, by four. Typhoid fever is not attended by valvular disease, but by such an enfeeblement of the muscular walls that dilatation may ensue. In six cases the evidence pointed to the conclusion that the disease was congenital. You will conclude, therefore, that, though it is (1) pre-eminently necessary that you should carefully examine the condition of the heart in any patient who is suffering from, or who has suffered from rheumatic fever, it is important to do so also in (2) those who have been subject to slight forms of rheumatic pain, and that (3) there is a large remnant requiring careful exploration whose diseases

are not to be traced to any obviously rheumatic condition.

Excluding, then, those I have mentioned, what are we to look for as the most common causes inducing heart disease? I will briefly enumerate some of them: Over-exertion and muscular strain—alcoholic indulgence—syphilis—tuberculosis—the puerperal state—poisoning by phosphorus—lead poisoning—imperfect and improper nutrition—disease which involves the structures contiguous to the heart and pericardium—disease which induces venous engorgement of the lung, whence distension, dilatation, and hypertrophy of the right side of the heart.

You may, from a general consideration of this introductory chapter, obtain in some degree an answer to the question which you will probably propound:—Under what circumstances of symptoms and previous history is it necessary for me to make a physical examination of the heart-region? There is one aphorism which I would impress on you, however, which covers the whole ground. It is this, *that you have never made a complete examination of any patient, whatever be his ailment, unless you have estimated, as far as possible, the condition of his heart.*

We shall now consider the mode of doing this. We pursue the investigation through our senses of sight, touch, and hearing. We do not grope for one sign which, when found, shall be conclusive to us; but after we have obtained all the evidence presented to our senses, our logical faculty must discriminate and lead us to the truth. We work by no single method, but by a combination of methods and modes of thought.

LECTURE II.

INSPECTION.

Cyanosis temporary and permanent—Pathological causation—Chilling of finger-tips — Clubbing — Anæmia — Graves' Disease—Sub-icterus—Arcus senilis—Cardiac dyspnœa—Orthopnœa—Decubitus in pericarditis—Cheyne-Stokes' dyspnœa—Œdema—Venous turgescence—Venous pulsation—Visible arterial pulsation—Locomotive pulse—Apex-beat—Area of visible cardiac impulse.

You may take it as an aphorism that you can never make a satisfactory examination in suspected heart disease, unless your patient be stripped to the waist. Very valuable evidence is afforded by inspection. The first point you will probably note is the general *hue* of the surface.

We will suppose that there is (A) a marked blueness of the surface. You will elicit whether this is temporary, sometimes passing away altogether, or permanent, varying perhaps in intensity, but never quite disappearing. The temporary blueness will be associated, probably, with attacks of cardiac asthma, of which dyspnœa is the great feature. The permanent blueness may also depend on the same cause as the temporary—viz., undue fulness of the venous system. In such case you will find respiratory trouble—bronchitis or emphysema or both accompanying it—and it affords strong presumptive

evidence of dilatation of the right cavities of the heart.

There is a form of blueness dependent on malformation of the heart so special that it constitutes the chief sign of, and gives a name to, the affection. Such is blue disease, morbus cæruleus, or CYANOSIS. Here you find a deep discoloration, in some cases approaching a black, involving all the surface, but especially manifest in the lips and the mucous membrane of the mouth. The colour is persistent, but is deepened when breathing and the heart's action are quickened, or when cough comes on. You will scarcely find any difficulty in recognising this condition—the hue is so characteristic; it is more pronounced and more general than that which obtains in ordinary venous congestion. Moreover, you will elicit perhaps that the affection dated from birth. If so, the evidence is nearly conclusive, but, if not, the diagnosis is by no means set aside, for the discoloration may not be obvious until periods remote from birth. Most probably, however, your patient will be an infant or young child. Nearly half the cases of this affection die before they are a year old; two-thirds before they are two years old; and though a few instances are recorded in which adult life has been attained, they are very rare.

We may now inquire, what is the pathological signification of the phenomenon? As a matter of fact, in a case of cyanosis the chances are rather more than ten to one that there is an abnormal communication between the right and left cavities of the heart, either between the auricles, owing to patency of the foramen ovale or between the ventricles, owing to imperfection of the inter-ventricular septum. Furthermore, the

chances are about six to one that the pulmonary artery is obstructed. The cause of the blueness, according to John Hunter, was the admixture of venous and arterial blood in the circulation. This explanation seemed simple enough. Owing to the structural defect in the heart, the dark-coloured venous blood mixed with the arterial, and the resulting darkened compound was propelled through the systemic arteries. This theory has been opposed in modern days, but it appears to me that its opponents try to prove too much. At any rate they hold that the explanation given above is *not* the true one of the cyanosis. According to them, cyanosis is due to congestion of the venous system, and this congestion is the result of obstruction of the pulmonary artery, or of some other malformation which induces an obstacle to the return of blood from the systemic veins. Premising that, in my opinion, the truth lies between these two theories, we will examine each of them.

The theory that the blueness results from direct mixture of venous with arterial blood, at first sight appears very plausible. Out of one hundred and ninety-five cases recorded by Stillé and Peacock, one hundred and seventy-eight presented abnormal communication between the right and left sides of the heart; admixture of venous and arterial blood therefore is possible and likely, and often inevitable. The hue of the patients is just that of those in whom venous blood is circulating: you see it in cases of impending suffocation; you may have many opportunities of witnessing it during the administration of nitrous oxide gas, when the blood is rendered of the venous colour by the excess of carbonic acid which it cannot get rid of. What are, then, the objections to the theory?

The first objection is afforded by the anatomical exceptions. Seventeen of the cases recorded presented no possibility of the arterial and venous currents abnormally commingling. This is sufficient to prove that the cyanotic tint cannot be *always* due to the arterio-venous anomaly. The second objection is that we can have communication between right and left heart without the appearance of cyanosis. This does not seem to me a fatal objection. The question is one of degree; the tint of the arterial blood may mask that of the intermixed venous blood, or *vice versâ*. Nay, more, a patient having this anomaly may present the peculiar coloration at one time and not at another. You may understand this by observing the differences in hue of a woman in health and in a state of anæmia and chlorosis. Let the blood corpuscles be numerous and healthy, and they would mask the coloration due to venous admixture, but let them be by any cause diminished in number or in colour, and the dark tint would declare itself.

Now let us turn to the other theory—that the coloration is alone due to venous congestion. The anatomical argument weighing with those who uphold this theory is, that obstruction of the pulmonary artery is an important and frequently-observed condition in cyanosis even when there is communication between right and left heart. But by their own data it is shown that this sign is less constant than the arterio-venous communication, the chances of the first condition being about six to one, the chances of the second more than ten to one. Moreover, it is an ascertained fact that there may be great obstruction of the pulmonary artery without cyanosis. A case under my care at the North-Eastern Hospital for Children

showed this most positively. A little girl, aged eight years and a half, presented signs of extreme anæmia; there was no blueness, but great pallor; all over the heart region was heard an extremely loud murmur with the first sound; it was loudest at the base of the heart. At the autopsy we found that there was obstruction by narrowing of the pulmonary artery. This was the only morbid condition to account for the murmur. It is evident, then, that there can be great obstruction of the pulmonary artery without cyanosis. It is, however, undoubtedly true that in some cases of blue disease, there has been found no communication between right and left heart, but only an obstruction upon the venous side which has given rise to a general congestion of the veins of the system.

From these considerations I think we are justified in arriving at these conclusions—first, that in a large number of instances of congenital cyanosis there is abnormal communication between right and left heart, and it is scarcely reasonable to doubt that the circulation of venous blood with the arterial tends to produce the peculiar blue coloration; secondly, that in some cases the blueness is produced only by undue fulness of the superficial veins. We know that such blueness can be thus produced, because we see it during attacks of dyspnœa, which spring from many causes, and we are familiar with it when severe cold affects the surface of the body. In this latter condition the cold causes contraction of the arterioles, and the blood is retained in the capillaries and venous radicles.

Having recognised your case as one of congenital cyanosis, you are by no means to stop here, but to

examine by all the methods which will be detailed hereafter, in order to determine, as far as possible, whether there be any cardiac malformation, and if so, what is its nature. For cyanosis may occur from causes which affect not the heart, but the lungs. Any cause which considerably impedes access of air to the lungs or seriously diminishes the extent of breathing surface, may induce cyanosis. You must, therefore, in these cases, carefully investigate the pulmonary as well as the cardiac conditions.

There is one point in cases of venous obstruction from any cause which may be classed under minutiæ, but which is of considerable diagnostic importance—the condition of the finger-ends. Notice if the finger-nails are blue in colour. If they are persistently blue, you may conclude that there is persistent fulness of the venous system. If they are occasionally blue, you will find the blueness coincident with attacks of dyspnœa. Notice also the temperature of the finger-ends so far as it is manifest to touch. Coldness means great defect of circulating power. Blueness and coldness together constitute a measure of the danger of attacks of cardiac asthma, and when these signs are persistent in cardiac disease, they show that the end is not far off. Notice next the *shape* of the finger-ends. When the return of blood from the veins to the right heart is obstructed to a considerable degree and for a protracted period, the finger-ends become thickened at their extremity, or *clubbed*. Dr. Dobell considers that when there is symmetrical clubbing of the finger-ends, and the nails are of the normal shape, the chances are in favour of heart disease; when the clubbing is attended with curvature of the nails over the ball

formed by the finger-tips, the chances are in favour of phthisis. The reason, probably, is that in the one case the adipose tissue exists in its normal quantity, and in the latter case it has wasted, just as in phthisis all the adipose structures waste.

We will now turn to another branch, and suppose that our patient does not present blueness but (B) pallor. The skin, the mucous membrane of the lips, the gums, and the conjunctival surface of the eyelids are pale; the sclerotic portion of the eyeball is of a pearly white. The patient, you say, is bloodless—anæmic. Anæmia may simulate heart disease, may be produced by heart disease, may aggravate heart disease. The method of differential diagnosis between the heart disturbances induced by anæmia and by organic heart disease respectively, we shall hereafter consider, but there are two conditions of anæmia which we may with advantage notice here.

The first is the condition of pallor associated with renal disease. We have seen in our introductory lecture that there is an especial relation subsisting between cardiac and renal disease. It is most important to discover such co-existence. If, in addition to pallor, you notice a "puffing" beneath the eyelids, or other signs of œdema about the face, you may suspect renal complication, and prepare (1) to examine the urine, (2) to use the ophthalmoscope. You will determine the specific gravity of the urine, ascertain whether it is albuminous and examine its sediment by the microscope. You will examine the fundus of the eye to ascertain whether certain changes which are known to co-exist with albuminuria are present. The red field of the fundus oculi presents in albuminuric retinitis irregular, or star-shaped

black and white patches, often glistening and presenting a metallic lustre, with, sometimes, dots of apoplectic extravasation.

The next condition usually, if not always, associated with anæmia, which we shall consider, is known as Graves' or Basedow's disease. This peculiar affection, by a strange displacement of an adjective, has been called exophthalmic goitre. It is characterized by a triple sign; (1) prominence of the eyeballs; (2) enlargement of the thyroid body; (3) irritable action of the heart. The affection is met with much more commonly in women, and between the ages of twenty and forty. The prominence of the eyeballs (exophthalmos or proptosis) is readily distinguished. The globe seems to be pushed forward in varying degrees from what seems merely an unusual size of the eyeball to a most unnatural protrusion—to such an extent that the eyelids cannot cover the globe, and the cornea becomes dimmed by inflammatory changes. The thyroid enlargement varies much in degree. In the cases I have seen, it has not been symmetrical; of three cases the enlargement was chiefly of the right lobe in two. The swelling is elastic to the touch, and pulsation of the arteries is readily felt. Care must be taken to distinguish it from aneurism. Dr. Stokes has mentioned a case in which such a mistaken diagnosis was made, and a day actually fixed for the performance of the operation of deligation of the carotid artery. The beating of the heart is visible over a wide area; the action is very rapid, excited by the least emotional provocation, and often exceeding 120 per minute. This affection does not rank as a heart disease, but it may lead to hypertrophy and dila-

tation. It is really an affection of the sympathetic nerve.

This strange disorder may be induced by causes operating on the nervous system. Trousseau has recorded a case in which its three symptoms were developed in a single night from excessive mental emotion. Milner Fothergill also has mentioned an instance of its sudden occurrence after emotional shock. Dr. Lauder Brunton considers that the palpitation characteristic of the disease is due to direct stimulation of the accelerator cardiac nerves which proceed from the vaso-motor centre in the medulla oblongata, accompany the vertebral artery, and, after passing through the inferior cervical ganglion of the sympathetic, are supplied to the heart. Irritation of the inferior cervical ganglion would account for the cardiac excitement. The thyroid signs are explained by paralysis of the vaso-motor nerves of the thyroid, which are derived from the same ganglion. Such paralysis causes dilatation of the small arteries at the back of the eyeball; so the globe is pushed forwards, the degree of prominence varying with the amount of dilatation of the vessels. In some fatal cases, implication of the sympathetic nerve has been proved to demonstration. In one instance its ganglia in the neck were found atrophied almost to extinction. In another case, the middle and inferior cervical ganglia were enlarged, hardened, and infiltrated with granular matter, the sympathetic nerve itself being involved in the morbid change. (See "Diseases of the Heart," by Dr. Hayden, p. 1061.) More recently Dr. Shingleton Smith, of Bristol has described a very important case (*Medical Times and Gazette*, 1878), in which there was entire destruction, by disease, of the inferior cervical ganglion on the left side of the neck. The ganglion had become completely atrophied, "its place being occupied by an inert mass of calcareous matter and fibrous tissue."

We will now suppose that our patient presents neither blueness nor pallor, but (C) *a yellowish tinge* of the surface. Deep jaundice is not frequent in heart disease, but a slightly icteric hue of the face, with a more deeply-tinged conjunctiva where it covers the sclerotic, is not uncommon in the later stages of

valvular disease when passive congestion of the liver is one of the troubles. When you find your patient past the prime of life, the victim perhaps of alcoholism, with a generally dusky yellowness of the skin, but without jaundice, the surface of the body somewhat greasy to the touch, the muscles felt to be flabby and found to be weak, patches of dilated capillaries upon the face, and venous turgidity of the conjunctiva—when, moreover, you notice that breathlessness or faintness is produced on exertion—you may fear fatty degeneration of the heart. For the diagnosis of this affection one sign has been adduced especially, and has perhaps sometimes been too strongly relied upon—the presence of an *arcus senilis* in the cornea. This requires careful scrutiny, for there are arcus and arcus. Near the junction of cornea and sclerotic, where the former should be clear and transparent, you may see a circle, a semicircle, or a crescent of opaque whiteness. If this be well defined, and the rest of the cornea bright and translucent, it is, probably, no indication of serious internal degenerative change. The corneal opacity is probably due to fibrous proliferation or calcareous infiltration. But if the ring be ill-defined, rather yellowish than white, the rest of the cornea being slightly cloudy, you may consider that the chances of cardiac degeneration are formidable.

Having learnt the lessons to be deduced from the complexion and hue of our patient, we will next consider the points to be gained from observing his mode of breathing and the postures which he assumes. In this section we will consider first cardiac dyspnœa. As I have said in the introductory lecture, this symptom is especially provoked by exertion. Thus it differs

from dyspnœa of pulmonary origin. You may find your patient breathe easily enough if he has been still for a short time previously to your examination, but if you call upon him to walk briskly for a few minutes, or especially if he ascend a few stairs, breathlessness comes on. This dyspnœa is peculiar—there is no real obstruction either to inspiration or expiration, but the patient gasps restlessly, there is an instinctive craving for more air to oxygenate the sluggish blood in the lung; there is *air-hunger* as the Germans expressively term it. Such is the dyspnœa of the earlier stages of valvular imperfection; but in the later stages the symptoms may be far more distressing. The sign which especially distinguishes the dyspnœa of the later stages of cardiac disease is *orthopnœa*—the patient cannot lie down; perhaps can scarcely recline from the perfectly upright position: the whole mental energies seem bent upon the one task of getting air into the chest. For a description of the most extreme condition of cardiac dyspnœa, I will quote the graphic words of Hope. The patient, "incapable of lying down, is seen for weeks, and even for months together, either reclining in the semi-erect posture supported by pillows, or sitting with the trunk bent forwards and the elbows or forearms resting on the drawn-up knees. The latter position he assumes when attacked by a paroxysm of dyspnœa; sometimes, however, extending the arms against the bed on either side to afford a firmer fulcrum for the muscles of respiration. With eyes widely expanding and starting, eyebrows raised, nostrils dilated, a ghastly and haggard countenance, and the head thrown back at every inspiration, he casts around a hurried distracted look of horror, of anguish,

and of supplication; now imploring in plaintive moans or quick, broken accents and half-stifled voice, the assistance already often lavished in vain; now upbraiding the impotency of medicine, and now, in an agony of despair, drooping his head on his chest, and muttering a fervent invocation for death to put a period to his sufferings." When the conditions of cardiac dyspnœa have long continued, you may find a new condition established, more merciful, but of even more fatal augury — carbonic acid poisoning. The blood is deteriorated by the carbonic acid, of which the respiratory effort is powerless to disembarrass it. The patient is in a constant state of drowsiness, with difficulty aroused, but waking in distress every now and then when the instinctive craving for more air asserts itself. In the end coma supervenes.

Such are the modes of comportment in chronic diseases of the heart wherein dyspnœa is a feature. In some cases of *acute pericarditis* you observe no respiratory trouble. Your patient usually lies upon his back, sometimes desiring that his head and shoulders should be raised; if he turns, he prefers to lie upon his right side, because when on his left side there is not only more direct pressure upon the pericardium, but the liver tends by its weight in this position to press upon the heart. It is usual in pericarditis for a patient to be very unwilling to change his position; syncope may be easily induced by rough movements, and it is to be remembered that such syncope may be fatal. The expression of countenance in pericarditis is often that of anxiety and apprehension, and wandering of the mind is common. Dyspnœa, such, as we have described in relation with chronic heart disease, may occur in pericarditis, but

you must not look for it as a common symptom. It occurs when the fluid effused into the pericardial sac mechanically obstructs the action of the heart, or in cases where a less amount of effusion weakens an already weak heart.

The "reason why" of all the forms of cardiac dyspnœa is not very easy to trace. In some cases the desire for the upright position is probably due to the relief from the pressure that the diaphragm, impelled by the abdominal viscera, occasions upon the right ventricle which such position induces. In others, where there is fluid in the pericardium, this relief is enhanced by the gravitation of this fluid to the most dependent parts of the pericardial sac, and thus the easing of the heart-muscle from pressure.

A characteristic form of *rhythmical dyspnœa* is occasionally, though rarely, observed. In such cases there are alternating periods of arrest, and of excitement of respiration. In the former period the thorax is absolutely motionless, and the patient appears almost as if dead. Then a faint wave of inspiration is noticed, followed by other respiratory efforts shallow and slow. The succeeding respirations become gradually deeper and quicker, until the chest is agitated with severe dyspnœa; then, arrived at its maximum, the paroxysm abates, the retrocession being as gradual as the onset, and at the end there is a period during which breathing is in complete arrest. This form of dyspnœa has been called after those who first observed and described it *Cheyne-Stokes' respiration*. Dr. Stokes termed it "respiration of ascending and descending rhythm." The periods during which respiration is suspended usually last from a quarter of a minute to half-a-minute, whilst the periods of rise and fall of respiration are of about the same (or rather longer) duration. In some cases arrest has been for only ten seconds, and respiration for seventy-five. The greatest duration of periods which I have found recorded is in a Memoir by Prof. Sacchi ("Riv. Clin. di Bologna," Feb. 1877, pp. 33-47). Here the respiratory arrest

and the dyspnœa each lasted for two minutes. When this peculiar symptom was first observed, it was supposed to directly indicate a condition of disease of the heart, especially fatty degeneration; further observation showed, however, that such disease was by no means necessary for its production. Then it was thought to be causally associated with dilatation and loss of elasticity in the aorta. Now that attention has been more fully called to the symptom, however, a numerous array of cases shows that this association also cannot be upheld. The symptom has been observed in cases which at first sight seem very diverse, but which can, I think, be conveniently divided into three categories. 1. Cases attended with cerebral lesions—viz., cerebral hæmorrhage, tumours, uræmia, shock from surgical injury, alcoholism, acute renal disease, tubercular meningitis. 2. Cases attended with lesions of heart and great vessels—viz., fatty degeneration, pericarditis, atheromatous disease of aorta, aortic aneurism, valvular disease (double aortic with mitral insufficiency, mitral stenosis dilated aorta co-existing, aortic regurgitation and obstruction), sclerosis of coronary arteries. 3. Cases of certain acute febrile diseases—viz., diphtheria (Hütterbrenner), typhoid fever (Wharry). A large majority of the cases occur in the male sex; for the most part the age is over fifty, when degenerative diseases are common, the exceptions being in the acute diseases which I have noted. The sign is one of *extreme danger*; the cases, with very few exceptions, have been fatal. The question as to the causation of this peculiar rhythmical dyspnœa is a very difficult one. The most plausible theory was first propounded by Traube. He considered that the conditions giving rest to the symptom have one feature in common—they impair the due arterialization of the blood supply to the nerve centres. You will realize that this would be the effect either of the direct impairment of the heart muscle, or more indirectly of the cerebral lesions. As regards the latter, it is to be noted that the symptom seems to be allied with causes that induce compression in the neighbourhood of the *medulla oblongata* (Cf. Guttmann "Handbook of Physical Diagnosis," Sydenham Society's Translation, p. 55). According to Traube's theory, then, the inadequate arterialization of the blood lowers the irritability of the *cerebral centre which presides over the respiratory movements.* In physiological

conditions this centre is called into activity by the accumulation of carbonic acid in the blood; when, owing to conditions of disease, the irritability of the centre is materially lessened, it follows that a much greater than normal accumulation of carbonic-acid is necessary to rouse it to action. In a case, therefore, which manifests Cheyne-Stokes' respiration, the first thing which occurs is the establishment of a condition of impaired irritability of the respiratory centre (this by Traube's theory through mal-oxygenation); the long respiratory arrest gives time for the accumulation of carbonic-acid in excess in the blood; arrived at a certain maximum, this begins to stimulate, slowly and imperfectly at first, and afterwards in increasing degrees, the centre, so that it develops the respiratory efforts till they culminate in dyspnœa. Then, as the centre ceases to be stimulated, or becomes exhausted, dyspnœa again supervenes. To this theory Filehne has instituted objections, but these rather go to modify and amplify than to destroy it. He would ascribe the phenomena to impaired coördination between the *respiratory* and the *vaso-motor* centres, the former must be less excitable than the latter. His theory is thus expressed by Guttmann:—At the end of the respiratory pause there is a large disappearance of oxygen from the blood, carbonic acid has accumulated, the vaso-motor centre is thereby stimulated, and the arteries (the cerebral arteries amongst the rest) at once contract; this produces a gradually-increasing anæmia of the respiratory centre, and inspiration becomes more and more deep; this, however, supplies the wonted oxygen to the blood, the arterial spasm is relieved, the anæmia of the respiratory centre passes off, and with it the exaggerated impulse to respiration, and breathing once more becomes superficial. When the arterial spasm has entirely subsided, so that the respiratory centre is abundantly provided with decarbonized blood, the stage of apnœa, the pause, is reached, and lasts till, by the abstraction of oxygen from the blood, the irritation of the vaso-motor nervous centre is renewed, and the whole series of operations again gone through. That the arteries are strongly contracted is proved by the increase of the arterial tension and of the blood pressure, while the anæmic condition of the brain is demonstrated by the fact that in young children, at the end of each respiratory pause, immediately before the recommence-

ment of respiration, and also while inspiration is gaining in depth, the great fontanelle is depressed. And, further, this form of dyspnœa may invariably be arrested at the very commencement of each seizure by the inhalation of nitrite of amyl, which dilates the vessels (*loc. cit.*, p. 56). The occurrence under my care of a typical case has given me close opportunities of studying the phenomena which we are discussing. In my case the symptoms developed simultaneously with an attack of right hemiplegia and aphasia. The attack was proved to be due to disease of many branches of the cerebral arteries, especially the middle cerebral of the left side, and there was much disorganization of brain-tissue, the morbid changes approaching close to the medulla oblongata. The heart and aorta were perfectly healthy. In the first case recorded (by Dr Cheyne) there was, in like manner, right hemiplegia and aphasia. Dr. Broadbent has recorded a case in which there were the same paralytic lesions due to cerebral hæmorrhage. Mr. Frederick Treves has described a case in which the phenomenon supervened on the shock of an injury—comminuted fracture of the leg, amputation: in this instance, also, there was no cardiac lesion. It can scarcely be doubted, that at least in certain cases the symptoms can be developed by cerebral and not cardiac causes. The theory, therefore, which ascribes its cause to an induced anæmia of, or a defective circulation in, the respiratory nerve-centre, necessarily fails. In my case, the sphygmograph showed that both volume and tension in the arteries were good and above the normal (see Part II.). There is, however, but little difficulty in concluding that in these cases the respiratory nerve centre is *directly* influenced—that it suffers a paralytic lesion, and so its irritability is impaired. This could be accomplished by the contiguity of disease or by shock. The acute diseases—typhoid fever and diphtheria—in which the phenomenon has been noted, are both occasionally attended with cerebral implication and paralysis—in such cases where the symptom supervenes, it may not unreasonably be ascribed to a paresis of the respiratory centre. Still, it may be doubted whether, in some cases, the symptom may not be initiated by disease of the heart-muscle itself. A review of the recorded cases seems to afford no positive evidence that it can be so initiated, for those in which degeneration of heart has been described have been almost invariably associated with disease

of the great vessels. Dr. Bradbury has recorded a case associated with fatty degeneration of the heart, but here the occurrence of syncopal convulsions showed the implication of he central nervous system. It is evident that the phenomenon is by no means to be interpreted as a sign of fatty degeneration of the heart; and it is even improbable that such affection of the heart, apart from cerebral complications, can in any case induce the symptom. It may be questioned whether the rhythmic dyspnœa can be induced by reflex causes. Its occurrence in aortic disease (certainly its most frequent association), as well as in aortic aneurism, would render this probable. It is possible, however, that the aortic diseases is not causal: it may but be the indication of arterial disease elsewhere, and the *vera causa* of the phenomenon may be found in disease of the walls of the cerebral arterioles. As a practical point, in any case which manifests Cheyne-Stokes' respiration, you will bear in mind the probability of disease of the vascular walls, or of cerebral affection. I consider that the initial lesion is paresis of the respiratory centre, and though this paresis *may be* produced by reflex nerve influence, it is usually a direct exhaustion from cerebral causes. Once initiated, the explanation of the phenomena on the theory of Traube is complete. In my case the interesting fact was established that not only the respiratory, but the cardiac centre was interfered with. The sphygmograph gave a remarkable tracing, showing a rhythmical irregularity in volume, an ample cardiac revolution being always succeeded by one of about half its amplitude. It has frequently been urged that in Cheyne-Stokes' respiration the pulse is uninfluenced, but it is obvious that in the absence of sphygmographic evidence the observation is of little value. In my case the peculiarity was not detected by the finger.

Having observed the general hue, posture, and mode of breathing, you will notice whether there are dropsical swellings of the surface of the body, pitting with the pressure of the finger, or whether there is evidence of fluid in the abdomen. Particularly inquire in what situation the œdema first manifested itself. It is an almost invariable rule that the dropsy, which depends upon cardiac disease, commences at

the feet and gradually extends over the the lower extremities. Thus it is distinguished from dropsy dependent upon renal disease, which commences in the face. Let me urge you, however, to receive the evidence of patients, when interrogated as to the locality in which the swelling first appeared, with considerable caution. The swelling of the face may have been transient, and even entirely overlooked. I must reiterate the rule which I have given you before—always examine the urine for albumen. If you find albumen present, you must still proceed with your examination of the heart, for a cardiac complication may exist with the renal disease. If you do not find it, you must hesitate before coming to the conclusion that the dropsy is cardiac, for it may be due simply to debility and anæmia. When the patient presents the concurrence of dyspnœa on exertion with a swelling of the feet which pits upon pressure, there is a strong presumption of cardiac disease. Cardiac dropsy proceeds upwards from the more depending parts, involving, after the legs and thighs, the scrotum and the general areolar tissue of the body (anasarca). The serous cavities, the peritoneal or the pleural, are usually the last to be affected. In renal dropsy the face is generally pallid; in cardiac it is dusky, and the surface of the skin is often marked by ecchymosed patches. In this latter condition slight wounds may become serious sores, and the sores sometimes gangrenous. The most common cause of dropsy in heart disease is imperfection of the valves. Of such, the most certain and most direct is disease of the valve of the right side of the heart—the tricuspid. This disease, however, is comparatively rare; the most common

valvular imperfection which gives rise to dropsy is disease of the mitral valve. Aortic lesions, however, sometimes induce the complication. It may also occur in fatty degeneration of the heart when there is no valvular imperfection.

We now turn from the more general to the more particular points of observation. Of these, first notice the condition of the veins, especially of those at the root of the neck—the internal jugular and the external jugular. Notice if the veins are distended. We have already noticed venous turgescence in the consideration of Cyanosis, and have seen that it is a sign of distension of the right side of the heart. You observe this fulness of veins in conditions of asphyxia —you can produce it temporarily by holding your breath—you observe it in those who play forcibly upon wind instruments, or in patients during the efforts of paroxysmal cough. In these cases there are stasis of blood in the lung, accumulations in the venous channels, and retention in the right cardiac chambers. In a chronic form you may see the same thing in patients affected with emphysema of the lung; in these the right chambers, being in a lasting state of distension, become dilated. Turgescence of the superficial veins may be due, however, to valvular lesion of the right side—to disease of the tricuspid or the pulmonary valves. In cases of venous distension you may often note that the positions of the valves within the vessel are marked by a slight ring or knot; sometimes the veins of the neck may be seen to be varicose. Closely observe the veins to ascertain whether they pulsate. The phenomena of *venous pulsation*, though not very common, are of great interest and importance. A very slight pulsation at

the root of the neck may be no more than natural: a slight wave of pulsation in a more distended jugular, synchronous with the systole of the heart, is a sign that the right auricle is distended, and that the contracting ventricle communicates to it its impulse. Be careful to note that the pulsation which appears to be in the vein is really so. The impulse may be conveyed by the artery beneath. To determine this, place your finger on the vein and press the blood upwards for a short distance so as to empty a portion of the vessel; retain your finger thus for a short period; then, if there is distinct venous pulsation, you will see the vein fill from below by jets synchronous with the beats of the heart. In the case of arterial pulsation beneath the vein, the latter does not fill by jets, and you readily feel the strong pulsation of the artery. In some cases you may observe that the pulsation is double, at first slight, being followed by a second stronger impulse. This is caused by the contraction of a hypertrophied auricle communicated backwards upon the column of blood in the vein, the closely succeeding pulsation being due to the reflux current impelled through the imperfectly-closed orifice by the right ventricle. Venous pulse is more common and more marked on the right side, the communication through the innominate vein with the vena cava being in a more direct line than obtains in the case of the great veins of the left side. If you satisfy yourself that there exists venous pulsation, the observation is of high diagnostic import—it means that the tricuspid valve is faulty, and permits regurgitation into the right auricle.

But, perhaps, you perceive not venous, but *visible arterial pulsation*. You notice, we will suppose, that

the carotids pulsate forcibly. There may be a vibration over the sternal notch due to the pulse in the aorta. If you look at the situation of the brachial artery in the upper arm, along the inner border of the biceps muscle, you will notice a jerking movement of the vessel at each impulse of the heart. On causing your patient to flex the arm, the movement of the artery is rendered still more pronounced. The vessel is seen to curve outwards from the centre line of the arm with the contraction of the heart, and then quickly return to its first position. Such is the *locomotive pulse* described, in association with the lesion which causes it, by Sir D. Corrigan, and often called *Corrigan's pulse*. This phenomenon is of great importance, for it is significant of one of the most grave of valvular lesions; incompetency of the semilunar valves of the aorta permitting reflux of blood into the left ventricle. The ventricle, which in these cases is hypertrophied, contracts with an exalted force and drives the jet of blood into the arteries with a sharp and sudden stroke, but, immediately on the subsequent dilatation of the ventricle, the arterial current flows back, leaking through the imperfect valve, and the arteries become abnormally empty. The natural result of the forcible filling of the partially emptied tube is the jerking pulse. You may observe visible pulsation of arteries in cases of aneurism of hypertrophy of the left ventricle, and especially when the vessels are tortuous and hardened from atheroma, but this peculiar jerking pulse is characteristic of aortic regurgitation.*

We will now narrow our area of observation to the neighbourhood of the heart itself. First notice

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* Vide subsequent Lecture on "Palpation."

whether the beating of the heart causes vibration at any part of the chest-wall or not. The heart-impulse may not be visible for many causes—a thick layer of subcutaneous fat in your patient, an encroachment over the normal situation of the heart by a portion of emphysematous lung, any cause which displaces the heart from the thoracic parietes, or an enfeebled condition of the heart itself. The absence of apex-beat is only of value when taken with other signs, but it is to be noted. Suppose that the beating is visible in one of the intercostal spaces, it is advisable not only to take a mental note of its position, but to mark it upon the cuticular surface with a soft pencil, a spot of ink, or tincture of iodine, or, if you wish a more permanent record, the stain of a moistened point of nitrate of silver.

The apex, under normal conditions, beats between the fifth and sixth ribs. To determine the spot at which it ought to be evident, you can make one or two observations. (1) Draw a vertical line rather more than an inch internally to the nipple. Where this line intersects, the fifth intercostal space is the spot where the apex, in the average of healthy hearts, is evident. The nipple can be taken as a fixed point in cases of men: but not so in cases of women. Then (2) draw your vertical line two inches from the left edge of the sternum; where this intersects, the fifth intercostal space is the spot required. Slight variations from this point may be, however, not abnormal. In adult life the apex may approach within a quarter of an inch of the nipple-line, or may recede to two inches nearer the sternum. I think you may take it that in adults, an apex-beat, which is either in the nipple-line or outside it, is abnormal. In the case of children, I

am accustomed to draw my vertical line midway between the left edge of the sternum, and a line dropped from the anterior border of the axilla, an apex beat *outside* this line indicates an abnormality. Any deviation, however, from the transverse line of the fifth intercostal space is irregular or abnormal.

Many causes may produce displacement of the apex from its normal position, but such displacement is not usually discoverable by mere inspection. We shall, therefore, briefly consider most of these causes hereafter. The displacement chiefly obvious to inspection is *where the apex beats below and externally to the normal position*. This indicates hypertrophy or dilatation, or both combined, of the left ventricle. In hypertrophy the beating may be observed as far as the eighth intercostal space, or even lower, and it is distinct, strong, and defined. In dilatation it does not usually reach quite so low, and is more diffused and more obvious in the *lateral* direction. In hypertrophy of the right side of the heart there is sometimes marked visible pulsation in the space between the situation of the normal apex and the ensiform cartilage. Note particularly the area over which the visible pulsation is manifest. Usually it is simply a tap in the fifth interspace. In hypertrophy this area is enormously increased, especially in children, before the ribs have fully ossified and the cartilages have become firm. I have observed the vibration in an extreme case of cardiac hypertrophy to extend outwards as far as a line depending from the anterior border of the axilla downwards, to half way between ensiform cartilage and umbilicus, and to the right to a considerable distance beyond the right border of the sternum. In young people hypertrophy of the heart gives rise to a

distinct prominence of the superincumbent thoracic wall. In cases of considerable dilatation and hypertrophy, the movement of the chest-wall caused by the cardiac contractions may be seen to be undulatory. It is obvious that the motions of the muscles which contribute to form the heart are not synchronous. Pulsations may be seen over the situation of a dilated auricle right or left. We shall learn in the next lecture that enlarged and hypertrophied auricles can give rise to pulsations which differ in point of time from the ventricular contractions. This is one cause of the apparent undulation. Adhesions of the pericardium may increase the irregularity of the movement.

The area of visible impulse may, however, be considerably increased, although there be no hypertrophy; in nervous palpitation, in some cases of anæmia, in Graves' disease or in chorea, pulsation may be observed over a space the size of the palm of the hand. You will note, however, that in such cases, the apex-beat is *not displaced* from its normal position, nor is the chest-wall bulged outwards.

You will notice particularly whether the præcordial region is rendered unduly prominent. The bony chest may be made thus prominent by rickets. You will recognize this condition by the fact that other portions of the thorax present nodosities, bulgings, or irregularities. Carefully observe whether there is any spinal curvature, for this, by causing depression of the thorax posteriorly, may induce bulging in front. If you are satisfied that there is distinct prominence of the heart region you will notice whether there is pulsation over the area—if so, you have obtained another evidence of hypertrophy. If, however, pulsation is feeble or absent, and the inter-

costal spaces are rendered convex, the presumption is strong that there is effusion into the pericardium. If the intercostal spaces are retracted so as to be rendered concave when the heart contracts, there is a probability that pericarditis has at one time existed, and has resulted in adhesions of the pericardium. In some cases this retraction with the cardiac systole is manifest in the epigastrium to the left of the ensiform cartilage. This is due to the attachment of the base of the pericardium to the central tendon of the diaphragm.

We may now recapitulate briefly the evidence which we have obtained by inspection in relation to its diagnostic value. We have obtained evidence which indicates the highest point of probability in cases of (1) cardiac malformation, producing cyanosis, (2) Graves' disease, (3) distension of the right cavities, (4) incompetency of the auriculo-ventricular valve of the right side (tricuspid), (5) incompetency of the semilunar valves of the aorta, (6) hypertrophy of the heart, (7) dilatation of the chambers of the heart. We have gained a considerable amount of information leading to the diagnosis of (8) acute pericarditis in cases when effusion has taken place in sufficient amount to obviously distend the pericardium, and of (9) a past pericarditis when pericardial adhesions have taken place in any considerable degree. We have obtained valuable evidence, though in less degree, of (10) conjunction of renal with cardiac disease, (11) fatty degeneration of the heart, and (12) valvular lesions where they have given rise to symptoms of dyspnœa.

We next propose to exercise the sense of touch to form or confirm our diagnosis.

LECTURE III.

PALPATION.

The pulse—radial compared with cardiac—Rhythm—Effect of effort—Position of apex beat—Displacement—Topography of heart—Mechanism of auricles and ventricles—Chronometry of cardiac pulsations—Presystolic, epigastric, and hepatic pulsation—Pericardial friction fremitus—Thrill.

We have now to cause our perceptions to enter by the tips of the fingers; we gain our knowledge through the sense of touch.

We will first examine the *pulse*. It is better to do so now that the chest, neck, and upper extremities are exposed, in order that we may consider the radial beats in relation with any obvious pulsations in arteries elsewhere.

(*a.*) Notice whether there be any *peculiarity* in the radial artery. It may be rigid, unyielding, presenting irregular hard plates, as it were, embedded in its wall. This indicates *atheroma*, a degeneration or calcification of the coats of the vessel. Your patient, in this case, will be past the prime of life, for the condition is essentially senile. The pulse will be felt to be hard and strong, the left ventricle having become hypertrophied, and contracting forcibly to overcome the obstruction created by the inelastic arteries. The changes of the radial are indicative of like changes in many other arteries of the body. You may probably observe the temporal artery

tortuous to the eye, and rigid to the feel, its pulsations visible. Frequently there is a semblance of strength in the pulse without reality, the shock being created by a feeble wave of blood in a rigid tube; in such case you will find that the impulse at the apex of the heart is feeble; this indicates that the atheromatous condition of the arteries is associated with fatty degeneration of the heart.

(*b.*) Observe in the next place whether there is a difference in volume and force between the pulsations of the two radials. If so, examine the brachials; if these are equal, the difference is merely one of irregular distribution. If there is a decided difference in the pulses of the upper extremities, there is a probability of aneurism of one of the great vessels.

(*c.*) Eliminating the above conditions, we now examine the pulse in relation to rapidity, regularity, strength, and volume.

(1.) Notable *slowness* of the pulse is generally due rather to neurosal than to cardiac causes; it may occur, however, in a heart extremely weak from fatty degeneration. It may also accompany atheroma of the aorta, probably from mechanical irritation of the branches of the vagus. The pulse has been noted as low as 20–30, and even 8–9 per minute.

(2.) *Rapidity* of pulse is much more common. It may be found associated with pyrexial conditions in the early stage of pericarditis, before effusion to any amount has taken place in the sac. Very frequently, however, abnormal quickness of pulse is not noticed at any stage of pericarditis. Of very far greater significance, is a point noticed by Dr. Walshe—*sudden variation of the rate* of the pulse. In a case of pericarditis, a very slight movement of the body may

increase the pulse from 80–90 to 120–140. Unusual quickness of the pulse occurs in many emotional conditions, and you must not rely upon it, except in connexion with other signs. In lesions of the mitral valve the pulse is usually quicker than in those of the aortic.

(3.) *Force of the pulse.*—This is a point of great moment. The apparent force of the pulse is notably *increased* in two conditions—hypertrophy of the heart and regurgitation through the aortic valves. It is *diminished* in degeneration of the muscular fibre of the heart, in dilatation of the cavities, and in most of the valvular affections.

In hypertrophy we find the pulse strong, full, and incompressible. As you feel the radial pulse with one hand, place the other upon the thorax over the heart-region, and you will find a strong, heaving, prolonged cardiac impulse as an accompaniment. There is an expression of power, both about pulse and apex-beat, which is quite wanting in simply functional excitement of the heart. In the latter case the stroke is not sustained, but abrupt and brief.

The other cause of exalted force of pulse, aortic regurgitation, has already been indicated to us by signs which we have considered under "Inspection." The pulse is first jerking, then collapsing; the artery strikes the finger with a sudden blow.

Suppose now that, on the other hand, you find the pulse small and feeble, *it is a good rule to elevate the patient's arm vertically above his head, and observe the characters of the pulse in this position.* This will enable you to eliminate doubtful cases of aortic regurgitation in which the pulse has become temporarily feeble;

in the aortic regurgitant lesion the pulse will become intensified instead of enfeebled, its hammer-like character exalted, and the patient may complain of discomfort. In most of the other valvular lesions, aortic obstructive disease excepted, in which no influence probably will be detected, the pulse is enfeebled by the vertical position of the arm. In some cases of mitral disease, and in conditions wherein the heart-muscle is feeble, the radial pulse in this position may be quite extinguished. The same occurs, however, equally in anæmia. The pulse being perceptible, notice whether the vertical position induces irregularity, especially irregularity of *volume*. If so, there is a probability of disease of the mitral valve.

You should now, while still keeping the finger upon the radial pulse, examine with your other hand the situation of the heart's impulse. It is very important if you note that, though the heart's contraction is strong, the pulse at the wrist is small and weak. This suggests imperfection of the mitral valve, inducing either regurgitation or obstruction. In mitral regurgitation the current of blood which should, by the contraction of the ventricle, be forced into the aorta, and thence to the systemic arteries, is, by the leak in the mitral valve, in part diverted to the left auricle. The pulse, therefore, is not proportionate to the strength of the heart's contraction, but is enfeebled in ratio to the amount of blood lost to the arteries by regurgitation. In mitral obstruction, the pulse is weak because the blood reaches the left ventricle with difficulty, and the latter contracts on an insufficient amount. It is not often possible by mere observation of the pulse to differentiate between

obstructive and regurgitant lesions at the mitral orifice. The irregularity so often attributed to the pulse of mitral regurgitation I consider to indicate engorgement or dilatation of the right chambers of the heart. For arguments see Part II. In many cases of mitral regurgitation the pulse appears to be quite normal. This indicates either that only a small quantity of blood is lost to the systemic arteries by such regurgitation, or else that the left ventricle has become hypertrophied sufficiently, and contracts with adequate force, to compensate for the obstruction caused by the reflux into the auricle. If you notice the pulse to be persistently weak, the contractions of the heart feeble, and yet by examination you find no evidence of valvular disease, there is a strong probability that the heart-muscle is enfeebled by degeneration.

If you find that the pulse is feeble, although the hand, placed over the præcordium, detects tolerably strong pulsation, and yet evidence of valvular disease is absent, then it is very probable that the hypertrophy of the heart causing the strength of systole is in the *right* ventricle, and not in the left.

In all cases in which there is a morbid condition of the right chambers of the heart, the radial pulse tends to be weak. The supply to the left ventricle is from the lungs; if, from any defect in the right heart, the lungs are ill supplied with blood, the left ventricle is also insufficiently supplied, and thus the systemic arteries are imperfectly filled.

Occasionally you may find that the hand on the præcordial region is sensible of a contraction, which does not make itself evident by a pulse at the wrist—there is an *inefficacious systole*. The ventricular contraction is at such times too feeble to produce a sen-

sible pulse in the remote arteries. It may be found, however, that, though lost in the radial, the pulsation can be detected in the larger arteries nearer the heart, such as the carotids. This is but a less significant expression of the conditions we are about to consider —viz., *irregularity* and *intermittency*.

(4.) *Rhythm of the pulse.*—Carefully distinguish between *irregularity of volume* and *irregularity in time*. In the former case the pulse is felt to occur at equal intervals, but is fuller at one beat than at another. You are sensible that varying volumes of blood are transmitted by the various contractions of the ventricle. This condition is rendered more evident by elevation of the arm, and the observation may give you an important aid to diagnosis. It is almost pathognomonic of mitral regurgitation with yielding of the right chambers.

We will suppose that the pulsations are irregular in time. Irregularity and intermittency are conditions which differ only in degree, so we will here consider them together. In intermittency, the pause between the pulses is longer, there being an interval equal to that occupied by a pulsation. The first thing I have to urge upon you is to be very careful in giving this phenomenon its due weight in a diagnostic sense, and nomore. I am afraid that mischief has been done many times by a hasty opinion to the effect that a patient has heart-mischief, when this sign has been too exclusively relied on. Remember that irregularity or intermittency of heart's action, and consequently of pulse, may coincide with organic integrity of heart, and even with good health. The first question I would ask you to propound to yourselves is :—Does this irregularity co-exist with a fair

strength of impulse, as felt over the præcordium or otherwise?

Intermittency may be merely a constitutional peculiarity. It may be due, as Dr. B. W. Richardson has shown, to causes operating upon the general nervous system. It may be superinduced by strong emotions, by terror, anxiety, grief, pain, fatigue; it may occur in organic diseases of the brain. It may be temporarily caused by attacks of indigestion. In the absence, therefore, of a notable feebleness of heart-beat, and excluding other diagnostic signs, you are not to conclude from mere intermission of heart and pulse that organic cardiac disease exists.

If, however, notable feebleness of impulse co-exists with irregularity, and especially ineffectual systole, it is of serious diagnostic import. It is the sign, the very early sign it may be, of a strike on the part of the left ventricle. Too languid to contract from the ordinary stimulus of the blood with which the auricle by its single contraction supplies it, it waits until a second, or even a third, contraction of the auricle has supplied it with more. This is the condition which obtains in mitral regurgitation and in dilatation of the ventricle. It is pathognomonic of dilatation of the left ventricle, and indicates that the muscular fibres have become degenerated, and have lost their tonicity. Most commonly it is associated with mitral regurgitation, which is the frequent inducing cause of such dilatation. When you observe in a case of mitral disease that the heart's action assumes this character, note it as a sign of prognostic import. In the absence of signs of valvular disease, the observation is of importance, both from a diagnostic and a prognostic point of view. When you

find it in conjunction with senile changes—with feeble but diffused impulse, with atheromatous vessels, "arcus senilis" of cornea, &c., you may know that a heart at one time hypertrophied has become degenerated, that it will never recover its power, and that the patient trembles on the verge of life.

A further question I would ask you to propound to yourselves when you notice in any patient irregularity or intermittency of heart's action is:—What is the effect of effort upon this heart?

An irregularity which is merely neurosal is scarcely affected by effort; the pulse is quickened of course, but its irregularity is often diminished rather than increased. When the irregularity is due to cardiac imperfection, however, very slight effort, such as making your patient walk briskly for a minute or two up and down the room, notably increases such irregularity. When there is dilatation, palpitation usually precedes the more pronounced irregularity; but when degeneration has proceeded far, the halting action takes place without notable quickening.

Notice also whether effort on the part of your patient causes distress, and what is the form of such distress. Little or no discomfort occurs in neurosal irregularity. In dilatation you find palpitation and dyspnœa; in degeneration, faintness and dyspnœa; in certain rare cases the ventricular halt is accompanied by horrible sensations, and a fear of impending death. Such was noticed by Romberg in a case in which a tumour involved the vagus nerve.

We turn from the consideration of the pulse* to that of the cardiac area itself.

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* For further particulars as to the signs afforded by the pulse, see Part II.

We have noted in our inspection of the chest where the impulse of the apex of the heart should be manifest. We proceed to confirm or enlarge our observation by placing the hand over this region. Suppose that

(*a.*) *The apex-beat is feeble or indistinguishable.* Before recording this as a positive observation, let your patient sit up and lean well forward; an apex-beat may then become evident which was before undetected. If notable feebleness of the beat be associated with weakness of the pulse and the signs we have before recorded as pertaining to the affection, you have gone very far towards the diagnosis of fatty degeneration of the heart. In rare instances, such feebleness may be found to be due to pericardial effusion; in such case the postural change just noted will serve you in good stead as an additional means of diagnosis, for the tilting forwards of the body may render evident the apex pulsation in a situation above the normal, probably in the fourth intercostal space, the fluid in the pericardium having tilted the apex upwards to this level. Palpation is of high importance in thediagnosis of pericarditis with effusion. In the earlier stages of the affection you may find excited, diffused, and, sometimes, tumultuous action of the heart with the general signs of pyrexia; as effusion takes place the apex-beat is enfeebled and carried upwards and to the left of its normal position; then, if distension be extreme, the beat is no longer to be felt. You must, however, draw no hasty conclusion from the observation that the apex-beat is feeble or imperceptible to the touch. It may be thus because the heart is overlapped by emphysematous lung, or on account of a thick layer of subcutaneous

fat in your patient, or, even in health, in persons with deep chests. You may readily understand that in women with full breasts the apex-beat may not be perceptible to the touch. It is a fundamental rule, however, that you should always observe in cardiac diagnosis—*to fix the exact spot of the apex-beat.* If inspection and palpation fail to do this, you will proceed to determine it by auscultation, as we shall hereafter consider.

We will now suppose that you have been able to feel the apex-beat, and have compared its position with that which it should normally occupy, as we have determined in the Lecture on Inspection. You will find that

(*b.*) *The apex-beat is displaced from its normal position.* Such displacement may take place from disease of the neighbouring textures and organs. Pleuritic effusion in the left cavity of the thorax may push the heart completely to the right side, so that the impulse which is wanting on the left side is felt right of the sternum; effusion in the right thoracic cavity, on the other hand, may push the apex left of its normal position. Emphysema of the lung pushes the heart downwards and towards the epigastrium, and oftentimes in this disease you find an impulse below the ensiform cartilage; you must examine further, however, before concluding that this is the apex-beat, for in emphysema the right side of the heart is usually enlarged, and the impulse which you feel is caused by the contraction of the right ventricle, the tip of the left ventricle being outside the point of obvious pulsation. Another disease of the lung which may give rise to a singular displacement of the apex-beat is fibroid phthisis; in such condition, when it affects the left lung, you may find

the chest-wall drawn inwards and the heart so displaced that its apex beats at or above the fourth rib. Tumours, aneurism, or cancer occupying the thoracic cavity may also cause displacement of the heart: enlargement of the left lobe of the liver, cysts, abdominal tumours, and dropsy, may produce a like result. We have already said that pericardial effusion tilts the apex upwards. All these causes must be eliminated by careful examination.

In the rare cases of transposition of the viscera, the apex of the heart is found to beat under the right, instead of under the left, nipple. The liver dulness then occurs on the left, and not on the right, side, whilst the stomach is to be recognized on the right.

We come now to causes of displacement intrinsic to the heart itself.

When the heart-apex is found to beat below and to the left of its normal position (fifth interspace and two inches from left border of sternum, or about an inch right of a vertical line through the nipple), you may diagnose hypertrophy of the muscular wall of the left ventricle or dilatation of the ventricular cavity. The sensations communicated to your fingers aid you to differentiate those two conditions. Remember that *hypertrophy* means *power*, and *dilatation weakness*, but the phenomena and their causes may be variously combined. A full, long, and heaving stroke is characteristic of hypertrophy; an excited, short, diffused, struggling beat indicates dilatation. Action with power is shown in the one case, excitement without power in the other. In hypertrophy and dilatation, the apex may be felt two or three inches, or even more, outside the nipple-line, and as low as the seventh or eighth intercostal space. If the apex happen to beat against a rib the shock is, of course,

subdued; it is necessary to recollect this, otherwise you might put down as feeble, an impulse which is really strong.

We now consider not only the *situation* of the tactile pulsation, but the *extent* of area over which it is manifest. Normally, as I have said, only a tap in the fifth interspace is felt; pulsation may be evident, however, over a superficial inch in strictly normal conditions. Pulsations in other positions may possibly be evident in health. Such may be felt in slight degree in the fourth intercostal space; or, and this more commonly, in the epigastrium to the left of the ensiform cartilage. By pushing the fingers upwards beneath the false ribs on the left side, you may sometimes feel the throb of the right ventricle. You must give these contingencies due weight, but, as a general rule, any pulsation manifest in other situations than the neighbourhood of the normal apex, is evidence of disease of the heart or the great vessels. We proceed to consider the conditions wherein

(*c.*) *Pulsations, apart from the apex-beat, are manifested over the cardiac area.* To appreciate these we should endeavour to obtain an idea of the normal TOPOGRAPHY OF THE HEART. The area occupied by the healthy heart may be thus roughly illustrated on the thoracic wall:—Draw a line, a little externally to the right border of the sternum from the second intercostal space to a point just below the fifth sterno-costal articulation. Draw a second line from a point just below the second sterno-costal articulation on the left side to the situation of the normal apex. Unite the extremities of these lines respectively above and below, so as to describe a quadrilateral figure. Of this superficies (which will be bounded above by the aorta which

crosses from right to left, and below by the diaphragm) about four-fifths are occupied by the right ventricle, which lies immediately behind the sternum and the third, fourth, and fifth costal cartilages of the left side, and culminates in the pulmonary artery at the second (left) interspace close to the sternum. The left ventricle is chiefly posterior; but it borders the right ventricle to the left from the third sterno-costal articulation to the apex, which itself constitutes. The right auricle is in the third interspace (right) and behind the third and fourth cartilages. The left auricle is for the most part overlapped by the pulmonary artery, but a small part of it is situated superficially in the second interspace left of the sternum.

If you find a forcible impulse of the heart towards the left of the border we have sketched as the normal left boundary of the cardiac area, the apex strongly defined in its beat and manifest below and to the left of its usual place, the pulse being full and strong, you may conclude that there is hypertrophy of the *left* ventricle. If you find a strong impulse in the epigastrium extending from the normal apex to the right of the ensiform cartilage and sternum, and yet a much weaker pulse than you would think such a systole would produce, you probably have hypertrophy of the *right* ventricle. Tuck your fingers under the false ribs to the left of the ensiform cartilage, and you will feel the contraction of the ventricular wall. We shall return hereafter to epigastric pulsations.

As I have before said, hypertrophy and dilatation are often combined. When the left ventricle is dilated as well as hypertrophied, palpation gives you a less localized and less firm and strong impression.

The impulse is more diffuse, the apex feels less pointed, and more rounded or globular to the finger.

When the left ventricle is dilated, but not hypertrophied, you feel its impulse over a wide area in the situation indicated, but it is felt as a short excited "slap."

Hypertrophy of the right ventricle scarcely ever exists without dilatation.

It has been supposed that auricular hypertrophy is not to be demonstrated by physical signs. I shall presently show you that this is not the case, for I have in many instances been able to demonstrate upon the surface of the chest-wall the contraction both of the right and of the left auricle.

When you have a general dilatation, or hypertrophy and dilatation combined, of all the cavities of the heart, the hand and the eye both perceive a fluctuation—or, as Dr. Walshe has expressed it, a seeming undulation—over the extended area over which the contractions of the cardiac chambers are manifest. This is obviously due to the fact that the contractions of the muscular walls of the various chambers are occurring not simultaneously, but in successive moments of time.

We will briefly consider the mechanism of the muscular walls of the heart. We should remember that the auricles and ventricles possess the double function of *reservoirs* and *propellers*. Such function is exercised in alternation—at rest they are reservoirs, in action they are propellers. You know that the rhythm of the heart comprises a period of contraction (systole), and a period of rest and dilatation (diastole). The latter occupies much the longer time: as a rule, the systole occupies one-fifth of the period; the

diastole, of course, the remaining four-fifths. During the period of repose, what is taking place? On the right side of the heart, the great veins of the body, the *superior vena cava*, and the *inferior vena cava*, are pouring their impure venous blood into the right auricle, which is gradually filling; the ventricle is becoming filled at the same time, for, the auriculo-ventricular valves being now flaccid, auricle and ventricle form one cavity. On the left side of the heart, the pulmonary veins are carrying the pure blood, which has been aërated in the lungs, to the left auricle, and hence to the ventricle, these cavities becoming filled simultaneously with the right. During all this period, the muscle of auricles and ventricles is receiving its nutrient supply, for the heart differs from all the other structures of the body in that, whilst the latter are supplied with blood by the heart's contraction, itself receives its arterial supply during its own period of repose. The muscle of the heart is supplied with blood through the coronary arteries, which arise from the aorta (pouches of Valsalva) just above the semi-lunar valves. The open mouths of these arteries had just been occluded by the flaps of the valves whilst the aortic orifice was open, but now in diastole, the valves falling back from the weight of the superincumbent column of blood and closing the aortic aperture, the channels are opened for the sudden in-rush of the stream of arterial blood into the tissue of the heart.

The cavities having become replete, the contraction of the heart, the systole, begins. The important point to notice is, that this contraction is not synchronous throughout all the muscular cavities of the heart; but that *the auricles always contract before the ventricles.*

You are aware that the fibres which constitute the muscle of the heart are under the control of the minute ganglia of the sympathetic nerve; the action of these ganglia is, however, controlled and co-ordinated by the vagus nerve, which restrains contraction until the movement shall be uniform and regular. The necessary stimulus to the contraction of the auricle is *distension;* until it is sufficiently filled with blood, its systole does not occur. Distension, then, provokes auricular contraction, and the ventricle which has already been filling by the passive flow of blood into it, is now more completely gorged by the additional blood forced into it by the auricle. In the normal heart, this precedence in action of the auricle is only momentary, the wave of contraction speedily ensuing in the ventricle. Still, it is of high importance to realize this priority of auricular contraction. Though the arrangement of the muscular fibres of the heart is such, that many of the fibres are common to all the cavities, yet the auricles are much more independent of the ventricles than the ventricles are of each other. Extremely early in fœtal life there is a mark of differentiation of the then single auricle from the single ventricle, but the right ventricle is formed by a doubling over of a part of the original (left) ventricle.

The necessary stimulus, then, for the contraction of the ventricles, is the extra repletion induced by the immediately preceding contraction of the auricles. The ventricular contraction, though strictly speaking vermicular, occurs in such a brief moment of time, that it is indistinguishable save as one movement in synchronism. The simultaneous contraction of the ventricles driving the blood into the aorta on the one

side, and the pulmonary artery on the other, the apex-beat, the pulse in the aorta, and the pulse in the pulmonary artery, are practically coincident in time.

I have said that in the normal state of the heart the pulsation of the auricles causes no apparent vibration of the thoracic wall; it is not so in disease, however; hypertrophy and dilatation of right or of left auricle may give rise to a visible pulse. As regards the *right* auricle, this has been generally admitted. The right side of the heart becomes dilated, and often hypertrophied, under all the circumstances which induce an undue repletion of the venous system. We have already alluded to these conditions, and we shall again briefly consider them in the next lecture. The right auricle is never hypertrophied and dilated without the right ventricle being in like condition. Under such circumstances you feel a heaving impulse, which seems to be very near the surface, extending from the normal apex across the epigastrium to the right side of the sternum; this is caused by the right ventricle, and you may feel a pulsation in the second or third intercostal space just right of the sternum, which is caused by the auricle. How to determine this we shall presently consider.

We turn to the *left auricle*. Undoubtedly, in certain cases, you may observe a pulsation in the second, third, or fourth intercostal space left of the sternum, but is this caused by the auricle? On this point there has been difference of opinion. Some writers have stated that inasmuch as the greater part of the auricle is covered by the great arteries emerging from the heart, and the muscular walls of the auricle are comparatively thin and feeble, its power of contracting with sufficient force to produce an impulse is improbable. These

observations are undoubtedly correct in reference to the left auricle when in a state of health. It is a very different matter, however, when it is in a state of disease. There is one condition of disease or malformation, which gives rise to great hypertrophy and dilatation of the left auricle—*narrowing (stenosis) of the auriculo-ventricular orifice of the left side (mitral)*. The causation of this hypertrophy and dilatation is easy to understand. I have explained that the contraction of the auricles is necessary to produce that perfect repletion (that additional supply to the already partially filled ventricle) which is required to call forth the ventricular systole. When the aperture between auricle and ventricle is so narrowed as to become an obstruction, the auricle has perforce to contract with enhanced power to overcome it. The auricle thus called upon for abnormal force of contraction becomes hypertrophied, and, from being in a state of preternatural distension, dilated. In the case of a boy of nine, who had constriction of the mitral orifice, I found at the post-mortem examination that the muscular wall of the left auricle was from $\frac{1}{8}$ to $\frac{1}{4}$ inch in thickness.* The normal thickness of the left auricle in adult life is somewhat more than $\frac{1}{12}$ inch.† According to Bouillaud, it is $\frac{3}{20}$ inch. Here was an auricle, the thickness of the muscular wall of which was in great part thicker than the thickest part of the normal adult right ventricle. Who could doubt the ability of such an auricle to communicate a distinct pulsation to the thoracic wall?

The foregoing considerations will point us to a plan

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* *Medical Times and Gazette*, January 10, 1874, p. 35.

† Flint, "Diseases of the Heart," 2nd edition, p. 21.

of demonstrating such pulsations as are caused by the auricles. A pulsation left of the sternum may be due to the contraction of the left ventricle, the pulse in the pulmonary artery, or the contraction of the auricle. A pulsation right of the sternum may be due to the contraction of the right ventricle or the right auricle, to the pulse in the aorta, or some aneurismal dilatation of that vessel. To determine whether the pulsations are, or are not auricular, we may adopt a simple plan of CHRONOMETRY OF PULSATIONS OCCURRING OVER THE CARDIAC AREA. It has been recommended that the movements of two doubtful points of pulsation should be compared by attaching to each, by means of a pellet of beeswax, a bristle carrying a small paper flag. The variation in time of the two movements is observed by the vibrations of the flag. The modification of this valuable plan which I adopt, is the following:—Cut two small circles of sticking plaster, about the size of fourpenny-pieces; transfix the centre of each by a pin, so that the head is in contact with the adhesive side of the plaster. Attach the one adhesive circle over the site of pulsation supposed to be auricular, and the other over the situation of the apex of the heart. The shafts and points of the pins projecting forwards, you have two levers which vibrate with the movements communicated to them by the several pulsations; these levers you elongate by attaching to them rolled "spills" of tissue paper. A still simpler plan, which I now generally adopt, is to pull out a small piece of cotton wool into the form of a tall cone and attach it by its base to the pulsating portion of chest-wall by means of a little ointment. Any number of these can be applied over points of pulsation, and the relation of their vibrations be thus compared.

If you have to do with an auricular pulsation you will see that the movement of its lever invariably *precedes* that of the lever adapted over the heart's apex. The movement is *presystolic*, and must be due to the contraction of the auricle. If the two contractions occur simultaneously they must be produced by the ventricle or in the pulmonary artery or aorta.

Pulsation of the pulmonary artery, which may be felt between the second and third ribs, close to the left border of the sternum, is only manifest when from any cause the left lung is retracted from the base of the heart. You feel that such pulsation is very superficial; it is said that even the click of the pulmonary semilunar valves may be felt as a little shock to the finger. It is interesting to note the relation of this phenomenon to respiration. If the lung be only partially retracted from the pulmonary artery, you will observe the pulsation to become more and more visible during *expiration*, whereas, during inspiration, as the lungs, becoming more and more filled with air, encroach over the heart, the pulsation becomes less and less evident till it ceases, reappearing at the next inspiration.

Pulsations of the aorta, which, of course, occur on the opposite—the right—side of the sternum, may be due to aneurism, or to displacement of the vessel which occasionally occurs in rickety chests.

It remains for us to consider *pulsation felt at the epigastrium.* We have already noticed the pulsation due to a displaced or hypertrophied and dilated right ventricle. Any considerable enlargement of the right side of the heart will give rise to a pulsation felt at the epigastrium. An impulse is sometimes communicated to the edge of the left lobe of the liver, which is to be felt

below the ensiform cartilage. Occasionally, but very rarely, the impulse is *reversed*: the integuments at the epigastrium are felt to be *retracted*, instead of propelled at each systole of the heart. In such cases there has been an extensive pericarditis, which has resulted in adhesion of the heart to the diaphragm and the liver.

But we have to consider pulsations felt at the epigastrium, which may be due to other causes, with a view to differential diagnosis. We will suppose that:

(1.) The pulsation is felt in the median line, that is, the line joining the ensiform cartilage and the umbilicus. It will probably occur to you that such pulsation may be due to aneurism of the abdominal aorta. The suggestion readily occurs, but though you feel a forcible pulsation over the vessel, you must hesitate very considerably before committing yourself to the opinion that it is due to an aneurism. Abdominal aneurism is rare. "So," as Sir William Jenner has said, "instead of being your first, it should be your last idea, that an abdominal pulsation is due to aneurism."* If there be an aneurism, you will feel a localized swelling of the vessel, which with, or just after, the systole of the heart expands equally in all directions, above, below, and laterally. A tumour, superficial to the aorta, may pulsate from the communicated impulse of the vessel; but then you will feel no lateral pulsation. You may find assistance in the diagnosis, by causing your patient to bend forward on the hands and knees: a tumour isolated from the vessel then recedes from it, and you no longer feel the pulsation, which in aneurism is unaffected by this position. Having eliminated abdo-

* Clinical Lecture on Tumours of Abdomen: *British Medical Journal*, 1869, p. 42.

minal aneurism and tumours to which the abdominal aorta may communicate its impulse, we will suppose that you yet find a pulsation in the central line, below the ensiform cartilage, which you have convinced yourself, by palpation under the false ribs, is not due to the impulse of the right ventricle. You will find such a pulsation very commonly; it is the pulse of the abdominal aorta. In the vast majority of abdominal pulsations there is no structural alteration to account for them; they are palpitations due to neurotic conditions. The abdominal aorta shows the excited pulse that the other great arteries of the body manifest. This phenomenon is, of course, most evident in spare people; you will find it in cases of anæmia, in dyspepsia, and specially the dyspepsia of old people whose arteries have commenced to degenerate. We will consider in the next place that:

(*B.*) The pulsation is felt to the *right* of the median line. I have said that the beating ventricle may propel the edge of the left lobe of the liver; an aneurism may do the same still more extensively; but in some cases it may be observed that *the whole liver pulsates.* This phenomenon is of rare occurrence, but it is of great diagnostic importance: it indicates regurgitation through the tricuspid orifice. The liver, in this condition, is like an erectile tumour; it pulsates with the systole of the heart. The right ventricle, by its contraction, instead of driving all the blood it contains into the pulmonary artery, on account of the imperfection of the tricuspid valve, forces some of its contents back again into the right auricle. Consequently, an impulse is given to the blood contained in the vessels opening into the auricle—*i.e.*, the great systemic veins and their tributaries. Hence the rhythmical injection of

the hepatic veins, and the consequent pulsation of the liver.* You will see that this phenomenon is exactly analogous to the venous pulse in the jugulars which we have before noticed. Both indicate the same morbid condition of the heart, tricuspid insufficiency. Suppose, now, that :

(*C.*) The pulsation is felt to the *left* of the median line. This may be due to an aneurism of the abdominal aorta, especially when it involves also the superior mesenteric artery. I once met with a case in which there was pulsation felt to the left of the middle line, midway between ensiform cartilage and umbilicus. The pulsation gave rise to distress, and was associated with a fulness at the epigastrium. The case had been diagnosed as one of cancer of the liver. Whilst, however, it was evident that the left edge of the liver encroached over the epigastrium, I could find no signs leading to the conclusion that there was any malignant disease. The left side pulsation was very difficult of explanation. I had the opportunity, however, of seeing the case several times, and I found (1) that the pulsation always became pronounced just previously to the catamenial period (the patient, a woman of full habit, was nearing the climacteric), and that it diminished almost to extinction, after the period was passed; (2) that it was always controlled, even to almost extinction, by large doses of quinine. The view that I took of the case was, that the pulsation was in the splenic artery. The patient completely recovered.

We have hitherto considered the evidence afforded by the well-known phenomenon, *pulsation*, as detected

* Aided perhaps by the pulsation of the inferior cava which lies behind the liver.

over the cardiac area. Now we have to consider other peculiar diagnostic signs recognizable to the touch.

THE SPECIAL TACTILE PHENOMENA MANIFEST OVER THE CARDIAC REGION are briefly, (*a*) friction, (*b*) thrill.

(*a.*) On placing the hand over the præcordium, you may, in certain cases, detect a sensation of rubbing accompanying the systole and diastole of the heart. You should cause the patient to hold his breath so as to convince yourself that the sensation is not communicated by any part of the respiratory mechanism. If you feel that the periods of contraction and dilatation of the heart are accompanied by this feeling of friction, you may be sure that your patient is suffering from pericarditis. The phenomenon is termed *pericardial friction-fremitus;* it is not common; in a very large proportion of cases of pericarditis it is not observed. There must be the concurrence of certain conditions to produce it. The pericarditis must be in an early stage; it occurs only at the commencement of effusion into the pericardial sac: the fluid effused must be thick or rich in fibrine; at autopsies in some cases of pericarditis, you may see the surface of the heart covered by a layer of material like soft butter, this is the kind of effusion which gives rise to the feeling of friction: the amount of fluid must be limited; as the pericardial sac becomes distended the exudation is less viscid, the heart is separated from the thoracic wall, and the systole is enfeebled—all these causes concur to prevent the sensation of pericardial fremitus: lastly, the heart must contract with sufficient force; a feeble heart does not produce it. This sign never occurs without the friction being recognizable by the ear, as we shall hereafter describe. You will find a great

number of cases of pericarditis occurring without this sign, but, where you observe it, it is of great importance; for it renders certain the diagnosis of pericarditis, and it enables you to state that the pericardial surfaces are roughened, or that the effused material is of a viscid character.

We come now to an interesting phenomenon, which will well repay careful investigation. This is:

(*b.*) Thrill. On placing the hand over the præcordium you are sensible of a peculiar vibration occurring over a certain area and at a certain period of the heart's action. The vibration is quite characteristic; a sense of trembling is communicated to the fingers—rapidly as if the finger touched the twanged string of a violin, or comparatively slowly as if the vibration had proceeded from the bass string of a violoncello. The French observers aptly compared the sensation to that experienced when one places the hand on the back of a purring cat or kitten; they gave to the phenomenon the name of *frémissement cataire*. It is characterized also by the terms *purring tremor*, and in German, *Katzenschnurren*. It is not sufficient, however, merely to note the existence of this sign. You must especially establish, (1) its position; (2) its rhythm.

You may feel it over the base of the heart, about the second intercostal space and right of the sternal border. Placing the tips of the fingers over the situation where the thrill is felt, now touch with the fingers of your other hand the spot where the heart's apex beats. This will enable you to determine the rhythm of the thrill. You find, we will say, that the thrill coincides in time with the apex-beat. Then you have either contraction (stenosis) of the aortic orifice

or aortic aneurism. You will notice whether the latter condition is indicated by a pulsatile swelling or the concurrent signs of aneurismal dilatation of the aorta. If not, you will proceed to confirm your diagnosis of aortic stenosis by auscultation and the means we shall hereafter describe. In rare cases the purring tremor may be felt over the situation of the pulmonary artery; it indicates obstruction of that vessel. Perhaps, however, you find that the thrill does not coincide with the beat of the apex but occurs during the diastole of the heart. A diastolic thrill is rare; it is characteristic of regurgitation through the aortic orifice. It signifies that the aortic valves cannot perfectly close, that they permit reflux of blood. So you may find this sign in conjunction with the phenomena we have noted as occurring in a like condition—forcible apex-beat and locomotive (Corrigan's) or water-hammer pulse.

Suppose, however, that you feel a thrill at or near the apex of the heart. In this position it is of the utmost importance to establish the rhythm of the thrill. I always advise that for this purpose you employ the fingers of each hand, as in the case of aortic thrill. First determine the spot at which the apex beats. You may find that this is also the point of greatest intensity of the thrill; to assure yourself of this, however, place a finger of your other hand a little externally. You will then convince yourself whether thrill and impulse occur together; in other words, whether the thrill is systolic. If so, if thrill = impulse, it is caused by regurgitation through the mitral orifice. Such a thrill is not common. You may find, however, and much more commonly, that the thrill felt in the neighbourhood of the apex

is not exactly simultaneous with the systole. You distinctly recognize that the finger which receives the impression of the thrill does so just before the finger of the other hand receives the impression of the impulse of the ventricle. The thrill is abruptly terminated by the ventricular systole—it is *presystolic*. Moreover, you may find that it is felt not just at the apex, but slightly internal to it, and at a higher level. This separation of thrill from apex-beat renders the detection of the rhythm of the former more easy by the rule which I have laid down. *Always investigate the phenomenon of thrill by using both hands, examining the site of the thrill by the finger or fingers of the one hand, and noting the apex-beat by the finger or fingers of the other.* It will be found that of thrills about the apex, that which is presystolic in rhythm is by far the more common. To recognize it is of very great importance. It is pathognomonic of contraction (stenosis) of the mitral orifice, and is due to the vibration caused by the forcible contraction of the auricle urging the blood through the obstructed outlet into the ventricle. We shall return to this subject when we come to auscultation, and consider the relation of thrill to murmur, but it is necessary to premise that both thrill and murmur are due to a like cause, but that we may have thrill without murmur and murmur without thrill. Vibration produces both phenomena; if the vibrations be insufficiently rapid they cannot be perceived by the ear, whilst they are obvious to touch; again, they may be so rapid as to be undetected by touch but detected by the ear; or the material which conducts the vibrations may be more suitable to convey impressions of touch in the one case, and of sound in the other. I wish strongly to insist that you shall give

due prominence to investigation by the sense of touch, and not fall into the error of esteeming auscultation the be-all and end-all of cardiac diagnosis. I have had cases in which the existence of thrill, presystolic in rhythm, led me to the diagnosis of mitral stenosis, though, in the one case, there was no murmur at all, and in the other it was so short as to be scarcely distinguishable. You must by no means draw any conclusion from the *absence* of thrill, but where you find a well-marked thrill before the impulse, you have, in my opinion, a certain sign of constriction of the mitral orifice.

We may thus summarize the diagnostic evidence to be obtained from the purring tremor. Felt over (*a*) the base of the heart its rhythm may be (1) systolic or (2) diastolic. If (1) systolic, it indicates (excluding aneurismal thrills, which are outside our subject, and the rare thrill denoting obstruction of the pulmonary artery) a morbid condition of the aortic valves which has resulted in a narrowing of the outlet from the ventricle. If (2) diastolic, it indicates regurgitation into the left ventricle, owing to imperfect aortic valves. Felt over (*b*) the apex of the heart, the thrill may be (1) systolic, or (2) presystolic.* Systolic thrill indicates regurgitation from the left ventricle through an imperfect mitral valve. Presystolic thrill indicates obstruction afforded by a narrowed mitral orifice to the current of blood issuing from the left auricle.

We will conclude the subject of palpation by a brief retrospect of the evidence which we have ob-

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* German observers call this presystolic thrill, *diastolic*.

tained. Excluding negative and doubtful evidence, you will observe that the sense of touch has given us signs of great and positive value in the following morbid conditions—atheromatous changes in the arterial vessels—hypertrophy of the left or the right ventricle; dilatation of either of the ventricles; pericarditis, in which it may have afforded evidence as to the stage of the disease, whether there is liquid effusion, whether the pericardium is roughened, or whether old disease has resulted in adhesions. Concerning valvular diseases of the heart and their effects, we may have received evidence of aortic obstruction from thrill; of aortic regurgitation from the locomotive pulsation of arteries, and, perhaps, from diastolic thrill; of mitral obstruction from presystolic thrill, as well as from pulsation of the left auricle; possibly of mitral regurgitation from systolic thrill; of tricuspid regurgitation from hepatic pulsation.

We shall next consider the evidence to be derived from percussion.

LECTURE IV.

PERCUSSION.

Increased resonance over heart—Increased dulness—Relations of areas of partial and complete dulness—Pericardial effusion—Cardiac hypertrophy and dilatation—Differential diagnosis of the two conditions.

THE object of percussion is to determine the size and shape of the heart and its relation to the neighbouring structures. In my own opinion the best plan of percussing the heart region is to use the fingers only. Place the fore and middle fingers of the left hand *closely* upon the surface of the chest, and tap with a short, decided, but not violent stroke, and in no hurried manner, with the ends of the fore and middle fingers of the right hand. I consider that the fingers of the left hand constitute the best *pleximeter*, and the fingers of the right the best *plessor*. Thus we obtain at the same time information by two distinct routes: the ear obtains evidence of *sound* at the moment that the tactile apparatus receives impressions of *vibration*.

Proceeding by the method of exclusion we will suppose that:

(*a*.) *The præcordial area is resonant*—that is to say, the percussion sound is clear and the vibration unimpaired—there is no dulness. The most common cause of this condition is emphysema of the lung. The morbidly dilated pulmonary air-cells encroach over

the area which, under ordinary circumstances, is occupied by the walls of the heart. Moreover, the heart may be pushed downwards by the too bulky lung. You may have been able, as I have indicated under "Palpation," to feel the beating of the right ventricle at the epigastrium.

A far less common cause of resonance over the heart-region is the presence of air in the cavity of the left pleura (pneumo-thorax). The tympanitic resonance in this condition exists, of course, over the whole of the affected side of the thorax.

In extremely rare cases, tympanitic resonance has been found only over the situation of the heart and pericardium; it indicates the presence of air or gas in the pericardium (pneumo-pericardium), a condition which may be induced by fistulous communication with the lung, the œsophagus, or the stomach, or by the decomposition of effused products of inflammation within the pericardial sac.

We will now suppose that:

(*b.*) *The præcordial area is dull.*—We shall have to exclude, first, cases wherein the dulness is not to be distinguished from that existing over the contiguous thorax. Pleuritic effusion in, or empyema of, the left side causes such dulness that the area of the heart cannot be mapped out by percussion. If such liquid effusion be in considerable amount, the heart may be pushed completely to the right side, so that its apex may be felt to beat under the right instead of under the left nipple. Condensations of the left lung, cancer, and abnormal growths in the thoracic cavity also may make it impossible for us to determine by percussion the outline of the heart. Assuming that these interfering circumstances have been eliminated,

we have now to consider the modifications of the percussion-note induced by the heart itself.

We have already considered the normal topography of the heart. We have now to remember that only a certain portion of the heart, covered by its pericardium, approaches close to the thoracic wall—a considerable part being overlapped by the borders of the lung. The portion of the heart which is uncovered by lung in conditions of health may be demonstrated with sufficient precision in the following manner:—Draw a vertical line through the centre of the sternum. Mark a point A, on this midsternal line at the level of the fourth left costal articulation. Note the point B, where the apex of the heart is felt to beat. Join A and B by an oblique line. Complete the right-angled triangle by drawing a line from the heart-apex, B, to the midsternal line at a point, C, just above the ensiform cartilage, at the lower part of the sixth costal articulation. The area enclosed by this triangle will be the portion of heart which, in conditions of health, is uncovered by lung. You should compare it with the area on the thoracic wall occupied by the whole heart as we have before described.

Now to percuss the heart-region. Begin by adapting the two fingers of your left hand held vertically, nails upwards (we are, of course, still supposing our patient to be in the vertical position, sitting or standing), to the thoracic surface, a little to the right of the right border of the sternum. After percussing in this situation, advance the fingers nearer to the midsternal line until the elicited sound is *dull*. Mark the vertical line where dulness commences by a soft pencil or by ink. In the next place commence to determine the left border of the dull area by percussing out-

side the point of the apex-beat. It is more convenient now to incline the fingers which are adapted to the chest-wall obliquely, pointing towards the sternum, as the left line of dulness will be oblique. Mark the line as previously. The upper limit of the dull area is determined by percussing from above downwards, the fingers adapted to the chest-wall being held horizontally. You have now obtained the upper and the two lateral limits. The lower limit is not so easily determined, because the cardiac merges into the hepatic dulness. The heart and pericardium are close to the left lobe of the liver, separated only by the diaphragm, and I hold that the dulness over these two organs cannot be discriminated. A line of dulness, however, can be determined between the apex on the left, and the commencement of liver-dulness on the right, and you should join these points by a line. For all practical purposes this procedure will indicate to you the area of complete dulness over the heart-region, the *superficial cardiac region* as it has been called, and it demonstrates *the portion of the heart which is uncovered by lung.*

But this is not all that is necessary. You should now reverse the order of your procedure, and starting from the line of dulness, make percussion farther and farther outwards until the sound has the perfectly clear character which it possesses over healthy lung. I do not think it necessary to overburden you with acoustic terms. To understand all that has been written about percussion—sonoriety, pitch, clang, *timbre*, intensity, tones, overtones, fundamentals, harmonics, &c.—one ought to be able at any moment to conduct an orchestra, tune a harp, read music at sight, and be equally versed in acoustics and prac-

tical medicine. Some observers have made assertions as regards the diagnostic power of percussion, that others cannot help thinking extravagant; thus, it has been said that it is possible to ascertain from the percussion-note alone whether a heart has undergone any fatty degeneration. It used to be told of Piorry, when I attended his class, that he was able, by knocking at the front door, to find out who was in the drawing-room, but such a critical ear is not given to everybody. Suffice it that the sound elicited by percussion over the heart in the situations where the lung overlaps it is modified by two causes—first, by the nearness of a dense body (the heart) to the point percussed, and secondly, by the existence between the point percussed and this dense body of an air-containing structure, the lung—*i.e.*, an arrangement capable of transmitting sonorous vibrations. As one proceeds outwards the sound becomes less muffled, and the vibration more manifest; but there is no *abrupt* line of demarcation to indicate the outline of the heart. Nevertheless, you should learn carefully to note where the sound and vibration are uncomplicated (that is, where there is lung vibration only), and where they commence to be modified. In estimating the curves of this area, I think it is best to employ as your pleximeter the little finger of the left hand, placed laterally against the thorax, instead of the fore and middle fingers. You should outline the area with ink or pencil; you will now have two concentric figures, the internal being the superficial cardiac area which we have before noted, the external the so-called "deep cardiac area" corresponding, if it be carefully, and, I think I may add, fortunately, ascertained, to the actual outline of the heart. For

practical purposes I would advise you to realize these two areas as, (1) *the area of dulness*, (2) *the area of deficient resonance*, and it is the mutual relations of these that you will find of diagnostic importance. The breadth of (1) the area of dulness in health and in adult life scarcely exceeds three inches transversely. As regards (2), the sound in percussing from above downwards begins to be impaired about the third rib: a curved outline can be mapped out at this level towards the right of the sternum, which indicates the aorta, but you cannot separate the aorta from the heart by percussion.

We will now assume that:

(*c*) *The præcordial dulness is extended upwards, and its outline is of pyramidal or pyriform shape.* Whenever you find that dulness extends upwards above the third rib, you have strong presumptive evidence of effusion into the pericardial sac. If the sac is distended with fluid you will find that the area of dulness extends from the articulation of the first or second costal cartilage, above to the sixth rib, or sixth intercostal space below.

Here let me insist on the great value of charts or diagrams as records of the physical signs of heart diseases. These may be roughly drawn in a few minutes, for sternum, clavicle, and ribs constitute almost all the outlines that it is necessary to depict. Be careful always to number the ribs as you outline them. Blank chest-charts have been prepared by many observers, and are extremely useful. You may procure one in a few seconds by a rubbing from the cover of this book.

Inspection and palpation have already indicated points which should be recorded on the chart. The

area of visible impulse should be outlined, the position of the apex-beat and the locality of thrill or tactile friction, if these exist, should be marked. Chalks, or pencils of different colours, may be used with advantage.

I have often found it of the greatest value to obtain graphic records from the chest-wall itself; for this purpose I use one of Perry's copying-ink pencils, which I have found most convenient. The plan is valuable as an *aide-mémoire* in all cases in which a physical examination of the heart is made. You first mark with the point of the pencil the spot of visible apex-beat; if this be extended, you denote the area of impulse by a dotted outline. Then you mark, in like manner, any other points of pulsation over the cardiac area, and proceed to further define the outline by palpation; afterwards the limits of præcordial dulness are traced out, and the positions and lines of conduction of murmurs, &c. The picture thus formed on the chest-wall itself gives you a vivid summary of the evidence which your physical examination has elicited. If you desire to preserve a tracing, you should also make a dotted outline of the position of certain of the costal cartilages, the ensiform cartilage, and any other fixed points that you may think necessary; you then apply over the portion of the chest thus marked, a piece of moistened tissue-paper. The paper, of course, takes the markings made by the copying-pencil, and you can thus preserve a permanent and exact record of the physical signs in the case, and can compare it at future times with tracings taken in like manner.

Now you will proceed to record the area of præcordial dulness. If this area be pyriform or pyramidal in shape, with apex upwards, the probability is strong that the case is one of pericarditis with effusion. We will consider the signs that are confirmatory of this view.

In the first place, the transition from dulness to lung resonance is *abrupt*. As I have said, the normal area of cardiac dulness widens gradually into pul-

monary resonance. The same occurs in many conditions of disease, but where you have the pericardial sac filled with fluid, the edge of lung, which normally overlaps the cardiac area, is pushed aside, and the area of deficient resonance (as opposed to dulness) is done away with. The line between pulmonary resonance and præcordial dulness is well defined, moreover the sense of resistance to the fingers on percussion is increased; vibrations are lessened.

In the second place, there may be noted a peculiar relation between the area of dulness and the position of the apex-beat. You may readily understand that if the area of dulness which you have mapped out be due not to liquid effusion but to enlargement of the heart, you will feel the apex-beat at the lowest limit of the dull area. Not so, however, when there is effusion into the sac of the pericardium. Then the apex-beat is tilted or floated upwards, and you get a certain breadth of dulness between the apex above and the stomach resonance below.

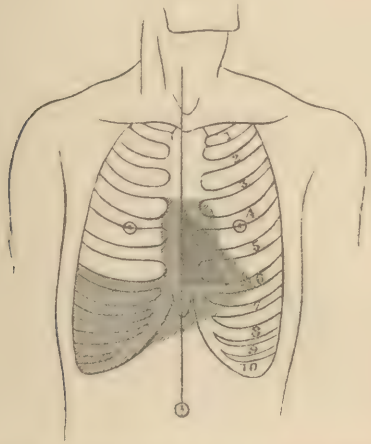
In the third place, you may observe that the extent of dulness varies within comparatively short periods of time. Effusion into the pericardium often takes place rapidly, and you may find that the dull area which you have mapped out previously, has, in a few hours, very considerably increased. From day to day there may be advances or recessions of dulness.

Again, in effusion into the pericardial sac the dulness varies with the position of the patient. On his lying down flat, the fluid subsides, then the apex-beat again becomes evident, and the percussion note becomes clearer, the lungs now returning to the position whence they were displaced by the fluid.

These signs, then, assure us that there is fluid in

the pericardium, whether due to pericarditis (the other signs of which you will search for) or to passive dropsy of the pericardium, which occurs, as I have

FIG. 1.



Showing the triangular area of normal cardiac dulness and its relation to the hepatic dulness. The shading indicates the area of deficient resonance—*i.e.*, the deep cardiac region.

said, with the manifestations of dropsy in other situations.

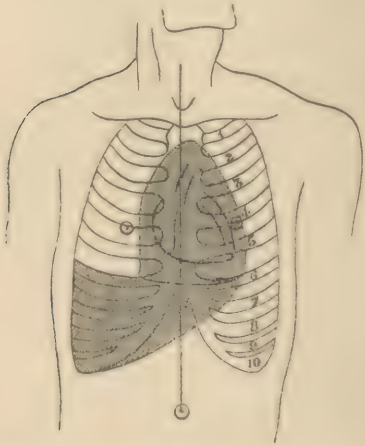
The foregoing points may, perhaps, be better understood by reference to the accompanying diagrams, for drawing which I have to thank Dr. Stephen Mackenzie.

Suppose now that:

(*d.*) *The præcordial areas of dulness and deficient resonance are extended laterally.* The impairment of resonance may be extended in either lateral direction.

Assume (1) that it continues to the *left* of the border of the normal triangle of cardiac dulness depicted in

Fig. 2.



Showing the pyramidal outline of dulness due to the effusion into the pericardium, and the relation of the heart to the distended sac.

Fig. 1. This extension of dulness must be due either to hypertrophy or to dilatation, or to both combined, of the left ventricle. In hypertrophy you will have already found that the apex beats below and perhaps outside its normal position. The area of dulness will be sharply defined, but it is only the extreme left of it which will be much changed in outline. The base line will be prolonged to the left of a line vertically drawn from the nipple, and it will incline downwards and towards the left instead of preserving its horizontal position.

In hypertrophy of the left ventricle, you will note that the præcordial dulness preserves its triangular form, but is prolonged towards the left side. You will of course notice the expression of power in the ventricular contraction, as evidenced to the eye, the hand, and the ear.

When the extension of dulness left of the normal area is due to dilatation of the left ventricle, the outline is less angular and more rounded than it is in the case of hypertrophy. The apex is less pointed and more globular. The left border of the region also is less defined; the area of deficient resonance is extended towards the left, and you will find the apex-beat less powerful and less sustained, with the character rather of a short and feeble slap.

As I have said before, hypertrophy and dilatation often co-exist. As a rule, the more obtuse the outline of the apex as obtained by percussion, the greater the probability of dilatation, but the further discrimination between these conditions we shall consider under "Auscultation."

We will suppose now the case that (2) the præcordial deficiency of resonance extends to the *right* of the normal area. We have not, in this case, to determine between hypertrophy and dilatation of the right chambers of the heart, because practically the two conditions always occur together.

You may (*a*) have no difficulty in discovering such right side hypertrophy. You may find that the area of absolute dulness extends beyond the right border of the sternum over the cartilages of the third, fourth, and fifth ribs, and when the right auricle is considerably dilated, you may find dulness extend from the second rib to the third intercostal space. In this case

the enlarged heart has displaced the lung which under other conditions overlaps it.

Often, however, (*b*) the determination by percussion of the outline of the right chambers is not easy. Sometimes percussion over the præcordial area elicits no dulness at all. In such case the heart is overlapped by emphysematous lung, and it is precisely in emphysema that dilatation of the right chambers is apt to occur. Disorders of the pulmonary circulation, involving as they do turgescence of the general venous system, lead up to dilatation of the right (the venous) chambers of the heart. Out of forty-five such cases of secondary dilatation of the right heart, Lancereaux observed twenty-four due to emphysema and chronic bronchitis, and six due to respiratory trouble induced by a malformation of the chest through rickets. Though the area of *absolute* dulness may be abolished in such cases, percussion, nevertheless, affords valuable data. You will be able to map out an area of *deficient resonance*. Commencing well to the right of the sternum, where the sound over the emphysematous lung is uncomplicated, you will advance nearer to the middle line of the sternum until you find that the sound, though not dull, is impaired. Thus you may obtain the outline of the deep cardiac area, and closely approximate to that of the heart, covered as it is by lung. You will, of course, observe the concurrent signs—pulsation of the right ventricle at the epigastrium, venous turgescence, perhaps venous pulse, and, if the symptoms are advanced, œdema spreading from the feet upwards.

We have now considered the evidence afforded by percussion of dilatation and hypertrophy of the left and right chambers respectively. You must, of course,

be aware that the condition of enlargement may affect both sides. In fact, dilatation of the right chambers may follow from impairment of the propulsive force of the left ventricle as consequence from cause. We shall see this especially when we come to consider valvular incompetence of the left heart. Suppose the left ventricle to be dilated and enfeebled, it is obvious that the first effect is a deficient propulsion of blood through the aorta—the general circulation is impaired owing to the reduced *vis à tergo*—the next result is engorgement of the venous channels; the right chambers of the heart may be considered as part and parcel of these venous channels—like the rest of the venous system, they are in a state of habitual plethora, so they become distended and enlarged, and their muscle becomes hypertrophied.

Having learnt the chief lessons to be derived from percussion of the præcordial area, we shall in the next lecture proceed to "AUSCULTATION."

BUREAU
APR 7 1881

LECTURE V.

AUSCULTATION.

PART I.

Immediate and intermediate auscultation — Stethoscopes—Normal heart sounds—Auscultation of voice to determine area occupied by heart—Topography of the valves—Modification of normal heart sounds—Accent—Exalted second sound, aortic and pulmonary—Strong pulmonary with weak aortic second sound—Skoda's sign of mitral lesion—Strong aortic second sound in renal disease—Prolonged first sound—Short loud first sound—Reduplication of sounds—Ineffectual systole.

I DO not think it necessary to call your attention to the importance of Auscultation. In the present day this is fully recognized. I am not sure that there is not a slight tendency towards an opposite danger—I mean a too exclusive reliance upon the signs offered to the sense of hearing. When the suggestion has come that the heart-region shall be examined, I have often observed that the stethoscope has been at once flourished, and the listening over the præcordium finished, the diagnosis has been supposed to be mature. I hope to have convinced you by the preceding lectures that precious aids to diagnosis are neglected if auscultation is alone relied upon.

On the other hand, the method of diagnosis cannot be complete *without* auscultation.

Auscultation may be direct and immediate, or indirect and mediate.

(*A.*) *Immediate Auscultation.*—You may obtain some general lessons by applying the ear directly to the heart-region, the chest being covered only by a fold of linen. By this you will learn whether the heart is displaced from its normal situation, and whether the sound of its contraction is strong or feeble. If prolonged and heaving you have corroborative evidence to add to the signs of *hypertrophy*, which have been deduced from previous examination. If feeble, you must hesitate before concluding that the heart-muscle is weak, for it is very probable that fatty tissue or emphysematous lung may intervene between the heart and your ear, and so occasion the feebleness of sound. If loud and heard over a wide area—a wider area than your examination by percussion of the space occupied by the heart would lead you to suppose—it is very probable that you have to deal only with *functional palpitation.*

(*B.*) *Intermediate Auscultation.*—Immediate auscultation is a useful preliminary; it gives you certain broad general impressions—but intermediate auscultation is absolutely indispensable. You have to differentiate sounds coming from situations very close one to the other. The valves of the heart all lie within a square half-inch of surface; if the tricuspid be excluded, portions of all may be covered by a superficial quarter of an inch. Practically, a sound obvious enough at a given point, may cease to be perceptible one-third of an inch therefrom. It is obvious, therefore, that the unaided ear cannot be relied upon, but that some means must be adopted for collecting and localizing sounds. The stethoscope must be used.

A few words as to the form of stethoscope most

suitable. The ordinary wooden stethoscope is the most generally useful; the thoracic end should be small enough to be adaptable to the intercostal spaces in thin subjects. As a rule, I think the metallic stethoscopes are inferior.

The binaural stethoscope is extremely valuable. For the cardiac auscultation of infants and children, it is indispensable. Not only does it shut off from the ears external sounds and allow the impressions to come only from the part at which its cup is applied, but its flexible collecting tubes allow each movement of the patient to be followed. Otherwise, children and infants may elude all your attempts at auscultation. Moreover, the sounds obtained through the double tube of this stethoscope, transmitted as they are to both ears, are perceived very intensely: sounds may be heard which otherwise would be inaudible. Still, I fully recognize that there is a danger—the sound may be too intense, so that if two occur near together, one may be drowned. The practical conclusion is, *use both forms of stethoscope*, the ordinary and the binaural. I have my own stethoscope so constructed that the same cup and stalk can be screwed into the ordinary wooden ear-piece, or into the junction of the tubes of the binaural arrangement.* So I examine successively with each form of instrument.

I have now for some time been accustomed to use a form of binaural stethoscope, introduced, I believe, by Professor Stern of Vienna. In this instrument the spring which keeps the terminal cups applied to

* This has been made for me by Messrs. Maw, Son, and Thompson.

the ears (and often when auscultation is long-continued, causes inconvenient or even painful pressure) is dispensed with. Each of the two india-rubber conducting tubes has a vulcanite extremity, which "plugs" into the auditory meatus, and with a little practice is easily retained. The distal extremity of each india-rubber tube plugs into a vulcanite cup, which is applied to the chest. Messrs. Maw and Son have made for me a modification of this instrument, with these advantages :—(1) the distal extremities fit into a short stethoscope made of vulcanite or wood, which, by adapting an ear-piece can be used as an ordinary stethoscope ; (2) the india-rubber tubing is graduated in inches, so that the stethoscope serves also as a cyrtometer, or chest measure.

Now it is necessary briefly to consider the NORMAL SOUNDS OF THE HEART. The mere application of the ear to the præcordium will convince you that the noise of the heart in action can be resolved into two sounds, which can be imitated by the syllables, *lubb-dup*. A little consideration will show you that the first, the longer sound of lower pitch, is separated from the shorter, sharper sound, by a short but distinctly appreciable interval of silence, and that, again, a longer pause or silence intervenes between the second sound and the recurrence of the first. Listening near the situation of the apex, you will find that the first sound co-exists with the impulse of the heart against the walls of the chest—that is to say, the contraction of the ventricles, the systole.

Without entering in the spirit of criticism upon the much-debated question of the exact mode of causation of these sounds of the heart, we shall find it sufficient for our present purpose to consider what is taking

place at the exact time at which these sounds respectively are occurring.

At the time of the production of the *first sound*, the following are the conditions. The ventricles are full of blood. The auricles have just contracted, impelling their contents through the auriculo-ventricular openings, and completing the replenishment of the ventricles. Then the muscle of the ventricular walls contracts—the tension of the enclosed blood forcing upwards the curtains of the auriculo-ventricular valves, putting them suddenly upon the stretch, and thus closing the orifices which they guard—driving the blood contained in the ventricles through the only now pervious openings—viz., the aorta on the left side, and pulmonary artery on the right.

At the time of the production of the *second sound* the conditions are these. The aorta, and through it the arterial channels of the whole body, as well as the pulmonary artery and its branches which carry venous blood to the lungs, have just been filled with a gush of blood. A momentary interval has occurred whilst these onward currents have passed, and during which the ventricles have commenced to become relaxed. The blood has been forced by the systole not into rigid tubes or into inert canals, but into the elastic arteries. When, therefore, the ventricles have ceased their contraction, and an appreciable pause has occurred during which the impetus of the blood-current has been unresisted, the semilunar valves of the aorta and pulmonary artery respectively are suddenly closed. The cause of such closure is twofold. First—the mere weight of the superincumbent column of blood in these great vessels; secondly—the elastic recoil of the previously stretched coats of the vessels of distri-

bution. In the case of the aorta, the elastic recoil comes not only from itself, but from every artery and arteriole in the whole system—in the case of the pulmonary artery, from the narrower limits of the distributing channels within the lungs. The second sound, then, is short, sharp, and sudden, and there is no doubt that it is due to the sudden closure of the semilunar valves of the pulmonary artery and the aorta.

During the pause following the second sound, the heart muscle is flaccid, and the cavities are becoming refilled.

The first sound, then, is coincident with the contraction of the ventricles; it is *systolic*. The second occurs at the moment of closure of the semilunar valves; it is called *diastolic*, but it is obvious that it occupies only a portion of the diastole. The diastole of the heart embraces all that period which is not occupied by systole.

Supposing the period of rhythm to be divided into ten equal parts, the following expresses, according to Dr. Walshe, the relative duration of sounds and silences:

First sound	=	·4
First silence	=	·1
Second sound	=	·2
Second silence	=	·3

In commencing the auscultation of the heart's area, you may have a duty as yet unfulfilled. Your former investigation may have failed to have indicated the situation of the heart's apex. This you must now determine by means of the stethoscope; observe and mark the extreme left of the situation at which the

impulse is heard at its maximum; that will correspond to the apex.

It may be necessary also to employ auscultation as a corroborative means of determining the area occupied by the heart, or by distended pericardium. You do this by auscultating the voice. The lines where vocal resonance terminates or becomes greatly diminished, indicate the border of the area.

We have now considered the necessary preliminaries for Auscultation of the Heart. The apex has been indicated, and the area occupied by the various chambers has been mapped out. We have already described the topography of the heart; we have now to consider the TOPOGRAPHY OF THE VALVES. The aortic, pulmonary, and tricuspid valves all are situated near the surface of thoracic wall—not so the mitral—this only approaches the surface at the point of the apex-beat, where the left ventricle comes towards the thoracic surface.

For the purposes of clinical investigation, the exact anatomical delimitation of the valves is of less importance than the determination of the areas on the thoracic surface to which the sounds proceeding from them are conducted. There are four centres where the sounds proceeding from the heart are determined to the thoracic surface:—

At the *heart's apex* are heard the sounds produced at the *mitral valve.*

At the *lower end of the sternum*, the base of the ensiform cartilage, the sound emanating from the *tricuspid valve.*

At the *second left intercostal space close to the sternum* the sound proceeding from the *pulmonary artery.*

At the junction of the second right costal cartilage

with the sternum and the contiguous second right intercostal space, the sound generated in the *aorta*.

As I have said, the area, as indicated by the sound, is not the absolute anatomical area.

1. "Sounds emanating from the *mitral* valve are not looked for directly over the valve (in the second left intercostal space, close to the sternal insertion of the third left costal cartilage), as here the latter lies behind air-containing lung tissue, a bad conductor of sound; they are auscultated rather at the apex of the heart, which is free of lung, and in immediate contact with the chest-wall, and towards which experience shows that sonorous vibrations coming from the mitral valve are transmitted with greatest intensity.

2. "In the same way the sounds of the *tricuspid* valve are not to be sought for exactly over their point of origin (behind the sternum at the level of a line drawn obliquely from the sternal insertion of the third left rib to the fifth right costo-sternal articulation), but somewhat lower down on the lower portion of the sternum."*

3. The sounds from the *pulmonary artery* are heard best over its exact anatomical position: from its origin in the right ventricle it rises to the lower border of the second left cartilage, where it divides into its right and left branches. The sounds from the vessel are, therefore, best heard in the second left interspace, where it is close to the thoracic wall.

4. "Sounds developed in the *aorta* are loudest, not just over the orifice of the vessel (in the second left intercostal space), but in the *second right intercostal*

* Guttmann, "Handbook of Physical Diagnosis:" New Sydenham Society's Translation. London, 1879.

space, in the direction of the ascending aorta. As the aorta at its origin completely covers the root of the pulmonary artery, the sounds produced in these vessels are necessarily intermingled, and would be indistinguishable from each other, were it not for the fact that those generated at the aortic valves are propagated most energetically in the direction taken by the current of blood in the ascending aorta, along the course of the vessel towards the second right interspace; at the latter point, therefore, aortic sounds should be auscultated."*

Deferring for a time any consideration of abnormal sounds heard over the heart's area, we will consider:

(*a.*) THAT THE HEART-SOUNDS ARE MODIFIED IN DEGREE.

A few words as regards *accent*. If you listen over the apex of the heart, you will find that the more pronounced of the two sounds is the first or systolic one. The accent falls on the sound produced by the contraction of the ventricles, such sound more readily reaching the ear. It would be expressed by the syllables, *lubb-dup*. At the base, however, the accent is reversed. The semilunar valves are nearer to the ear, the accent falls on the shorter second sound and the expression would be *dup-lubb*. This may give rise in some cases to a little perplexity: you may take the second sound for the first, and *vice versâ*. The difficulties may be obviated, however, by a very simple rule. When you are auscultating the base of the heart, place the finger over the spot of the apex-beat, or, if this be too feeble to be distinguished, over the

* Guttmann, *loc. cit.* p. 262.

situation of the great vessels at the root of the neck. The impulse or the pulse will thus tell you which is the first sound: of course if the sound you hear be synchronous with the systole or pulse, it is the first sound, if not it is the second.

It is well, in my opinion, to commence by auscultating at the base of the heart, because the situations of the aorta and pulmonary valves are fixed points, whilst the apex is variable.

At the base, in the absence of abnormal sounds, it is to the *second* sound that you must pay attention. The quality of the first sound will be investigated at the apex.

You will place your stethoscope over the second right costo-sternal articulation, then carry it across the sternum to the second left interspace and the articulation of the sternum with the third costal cartilage on the left side. You will observe and compare the qualities of the second sound in these right and left situations respectively. The aortic second sound is normally more pronounced than the pulmonary. It is the closure of the aortic valves which gives, for the most part, the character to the second sound which is heard over the general area of the heart—that is to say, the aortic overpowers the pulmonary second sound. Suppose now that (*a*) *the aortic second sound is intensified.* It is obvious that you must train your ear by observing the second sound in cases of health. When you have recognized its normal intensity only are you in a position to judge of its exaggeration. If on placing your stethoscope over the second right costo-sternal articulation you hear a sharp and loud flap coming closely to the ear, your inference must be that an abnormal amount of blood, which has been forced into the aorta by the contraction of the left ventricle, has, by the

energy of its reflux, closed and put on the stretch the semilunar valves. Carrying your inference further, you will find that you must have a strong, muscular, in other words, *hypertrophied left ventricle*, and an aorta capable of containing an unusual amount of blood, in other words, a *dilated aorta*. In conjunction, therefore, with other signs, you will find persisted exaggeration of the aortic second sound a valuable evidence of hypertrophy of the left ventricle. Conversely, if you find that (*b*) *the aortic second sound is feeble*, you may infer that there is a deficiency of arterial blood supplied to the aorta by the ventricle. This observation is sometimes valuable as a means of prognosis when the blood has been diminished in quantity by hæmorrhage, or when the tone of the heart has been enfeebled by disease. In fevers a very weak second sound is a bad prognostic. There are certain circumstances, however, which mar or modify such absolute deductions from the observed force or feebleness of the second sound. There may be a sharp and loud second sound when the aortic valves are unusually thin. Or it may have a dull or leathery character when the valves are thickened. You must, therefore, remember to record the observation, but to make no absolute deduction without comparison with other physical signs.

Suppose now that (*c*) *the pulmonary second sound is intensified*. This is a sign of more absolute importance. I have said that the great cause of intensification of the aortic second sound is an increase above the normal quantity of blood in the aorta. In case of the pulmonary second sound another cause is present to augment it. The pulmonary is a smaller circuit, and the blood contained in it, like the water in a Bramah press, exerts equal pressure in all directions. The

blood-pressure in the general systemic circulation is modified in various ways, by subsidiary circulations, as the portal, and by many special conditions in the various tissues. The pulmonary circulation may be looked on as if conducted by a single flexible and contractile tube from right ventricle to left auricle. If, from any cause, therefore, there is any obstruction to the flow of blood within the pulmonary circuit, the result is not only congestion of the lungs, but also "a uniform increase of the tension throughout the whole of the pulmonary circulation, often accompanied, if long continued, by slight dilatation of the pulmonary artery, and always by a closure of the semilunar valves with an exaggerated force proportionate to the hindrance it has met with."*

Of still greater importance than the estimation of the absolute strength or weakness of the aortic and pulmonic second sound, is the observation of the relative force of the sound in the two situations.

Suppose that you find that (*d*) *the pulmonary second sound is intensified whilst the aortic is enfeebled.* Your first inference will be, as I have just indicated, that there is exaggerated tension in the pulmonic circulation—that is the cause which must produce the exalted second sound. This may occur, however, in any condition of respiratory trouble when there is congestion of the lungs, but then the second sound over the aortic valves need not be perceptibly or persistently enfeebled. The co-existence of a strong pulmonary with a weak aortic second sound is a sign that whilst the pulmonary artery receives too much blood, the aorta receives too little. It is valuable evidence, as Skoda first pointed

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* Balfour.

out, of a diseased condition of the mitral orifice either permitting regurgitation, or inducing obstruction of the blood-stream. In the case of the former, the explanation of the phenomenon is easy: the arterial current, urged into the aorta by the contraction of the left ventricle, is diminished by the amount which regurgitates through the imperfectly-closed mitral orifice: the aorta, therefore, is insufficiently supplied with blood, and the sound caused by the reflux against its semilunar valves is consequently weak. In the case of mitral obstruction (stenosis) the effect is the same, though the cause is different. Here the blood reaches the ventricle with difficulty on account of the narrowing of the outlet from the auricle, systole takes place upon an insufficient amount, and the aorta is ill supplied. The case is just otherwise with regard to the pulmonary artery; as a consequence of the deficient arterial supply, induced by both the above conditions, there has been a reduction of the rate of venous return, and engorgement of the venous system, a secondary dilatation of the right side of the heart. In regurgitation, a backward current is created by the systole of the ventricle; in obstruction, the contraction of the auricle tends to cause reflux. All these causes, therefore, produce high tension in the pulmonary circuit. So, with the arterial anæmia there is venous and pulmonic hyperæmia, and hence exaltation of the shock of recoil of the semilunar valves pertaining to the right heart. This observation of the comparative intensification of the pulmonary second sound is valuable, not only as a sign of the existence of the conditions I have just mentioned, but as an indication of their degree—so it is of great importance in prognosis. In proportion to the comparative feebleness of the aortic second sound is the failure of the ventricle; in propor-

tion to its strength is the power of the ventricle, by its compensatory hypertrophy, to overcome the obstacles imposed.

If you find the opposite condition when (*e*) *the aortic second sound is relatively intensified*, it is evidence, of course, of hyperæmia on the arterial side of the circulation. I have already spoken of this as suggesting hypertrophy of the left ventricle, but it is only to be relied on in concurrence with other signs. There may be much hypertrophy of left ventricle concurrent with mitral regurgitation, when, as we have just seen, the aortic second sound is enfeebled. In such case the hypertrophy is insufficient to compensate for the loss by regurgitation through the mitral orifice. Again, though the left ventricle may be hypertrophied and the aorta dilated, the coats of the latter (through atheroma for instance) may be so inelastic that the second sound may evidence no exaltation. There is one condition of disease, however, in which accentuation of the aortic second sound is a sign of considerable importance—this is *Chronic Bright's Disease*. I have called your attention already to the conditions which exist in this disorder. There is a peripheral obstruction to the circulation of the blood through the terminal arterioles, coincidently there is heightened power of left ventricle; the necessary consequence is an abnormal excess of tension in the arterial system, and this state of tension occasions the sharp and pronounced aortic second sound.

It is important to note the relative predominance of the first and second sounds at the base of the heart. You may find that the first sound is nearly or quite inaudible, the *only* sound is the diastolic. In such case there is strong presumptive evidence of hypertrophy of the left ventricle.

We turn now from the base to the apex; and our

consideration will be devoted to the first instead of the second sound. Suppose we find that (*f*) *the first sound is abnormally prolonged.* We have then evidence of hypertrophy of the left ventricle. In auscultating at the apex I advise you to keep distinct in your minds the element of *sound* and the element of *duration.* You see that as evidence of hypertrophy I have called especial attention to the latter. The first sound is constituted by two factors, the contraction of the ventricular walls, and the tension of the auriculo-ventricular valves. The muscular sound of the contracting ventricles is dull and prolonged, whilst the sound of the stretching of the curtains of the valve is (as you know it to be in the case of the semilunar valves) sharp, short, and sudden. In proportion as the muscular element of the first sound preponderates over the valvular, so the first sound will be dull and prolonged.

In hypertrophy the first sound is dull, partly because the flap of the valve-curtains is less easily conducted to the ear through the thick muscle of the ventricular wall, partly because the contraction of the ventricle itself produces a dull (muscular) sound. It is prolonged, because the thicker the muscle, the longer its period of contraction. (For a review of the numerous theories as to the causation of the sounds of the heart, see Hayden's "Diseases of Heart and Aorta," 1875, pp. 93 *et seq.*) An able investigation on the question has been made by Guttmann (see "Handbook of Physical Diseases," translated by Dr. Napier: New Sydenham Society, 1879, p. 265, *et seq.*), who concludes thus:—"Although the whole question in dispute cannot yet be said to be definitely settled, the conclusion to which the present state of our knowledge seems to point is, that *the first sound is essentially of valvular origin, and only to a slight extent muscular.*"

If on the other hand (*g*) *the first sound is shortened*, it is evidence that the ventricle is weak. In this rela-

tion we may have several conditions. The first sound, though short, may be loud. It is rather a frequent mistake to interpret loudness of the first sound as a sign of increased power of the ventricle; as a rule it is the opposite. A loud, sharp, short, flapping first sound at the apex indicates dilatation, and consequent feebleness of the ventricular walls.* In such case the muscular sound is reduced to a minimum, and the ear appreciates in the greatest degree the valvular element of the first sound. Then the first sound approaches the second sound in character and duration. You may even be puzzled to discern the one from the other. The following rule will easily guide you: whilst you are listening by means of your stethoscope, place your finger over the spot nearest the heart apex, at which a decided pulsation is to be felt. If the apex-beat is evident, place your finger over its situation, if not, observe the carotid pulse (not the radial, for that is fallacious, because it is not always coincident with the cardiac systole). You thus have a certain indication of the time of the systole, and you will be able at once to know whether the sound which you hear is synchronous with it—*i.e.*, the first sound—or intermediate between pulse and pulse, when it will, of course, be the second sound. But the first sound may be short and yet feeble, and this is evidence of a still graver condition of heart-debility. You then have, not only an impaired muscular sound, but a weak valve sound, for the enfeebled muscle cannot create sufficient tension to produce the pronounced flap of the valvular curtains. In such

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* Such a first sound is met with in Graves' Disease. See p. 30.

case you must look for the other signs and symptoms of degeneration of the muscular walls of the heart. In extreme cases of fatty degeneration, the first sound may be lost altogether.

We will next consider briefly a condition which is sometimes more interesting than important, but which must always be noted and deciphered where it exists—(*h*) *the sounds of the heart are reduplicate.* Instead of the single sound of the normal rhythm you hear two sounds following as if the syllables *tah-ta* were uttered. This may occur at the base or at the apex, or in both situations. It may be heard as a permanent sign in conditions of health, as a transient occurrence in such conditions, as a permanent sign in disease, and lastly, as a phenomenon, continuing definitely during a morbid condition of the heart, and vanishing when health becomes restored. On what does it depend? The inquiry is interesting, but the answer is not easy. We will first take the case in which the phenomenon is manifest at the apex and not at the base. Now I must ask you carefully to discriminate between *reduplication* and *ineffectual systole.* I have already said that in some cases contraction of the ventricles may be repeated without producing a pulse to be felt at the wrist; so if you count the pulse at the wrist and compare with the rate of pulsations as determined by auscultating the apex, you find that the two records do not exactly correspond.

Many cases and observations have been recorded which illustrate this ineffectual working of the ventricle, notably by Von Dusch, Potain, Hayem, and Flint. This is not reduplication, however; in reduplication there is a repetition only of a portion (the systolic or diastolic sound as the case may be)

of the cardiac revolution, whilst in ineffectual systole the whole cardiac revolution is repeated once, twice, thrice, or even four times, until sufficient arterial tension is attained to produce the pulse. In such case the pulse may be sometimes felt in the larger vessels, as the carotids, before it is manifest in the radials. You may, perhaps, understand this better if I indicate it graphically. Let the syllables *tah-ta* express the cardiac sounds, *tah* being the first sound and *ta* the second. Then INEFFECTUAL SYSTOLE is thus represented:—

tah ta tah-ta = Pulse: or
tah ta tah-ta : *tah-ta* = Pulse: or
tah ta tah-ta : *tah-ta* TAH-TA = Pulse,

the latter indicating varying expressions of strength of the sounds.

REDUPLICATION OF THE FIRST SOUND is represented by

tah tah ta = Pulse: tah tah ta = Pulse, or
tah *tah* ta = Pulse: tah *tah* ta = Pulse.

A doubling of the first sound may be heard in health at that period of the respiratory rhythm when expiration has just been completed and inspiration is commencing.

According to Dr. Hayden the phenomenon may be associated (1) with simple functional derangement of the heart, such as nervous palpitation and fluttering, usually accompanied by anæmia; (2) with attenuation and weakness of the ventricles in persons of middle age of nervous temperament; (3) with a weak degenerating heart, and dilated atheromatous arteries; (4) with simple hypertrophy of the ventricle. According to Guttmann it may occur temporarily in perfectly healthy persons; it may be noted at times

in diseases of the heart—sometimes connected with mitral, sometimes with tricuspid disorder—but cannot be said to be characteristic of any particular affection.

As far as regards clinical diagnosis, therefore, you may conclude that the sign is of but little importance. Of course you will in every case which comes before you be careful to note it, because, as we shall see, its investigation may have a strong bearing upon the interpretation of some unsolved cardiac phenomena.

We will now consider reduplication of the second sound, which is much more commonly observed. This may be expressed by

tah ta ta = Pulse : *tah ta ta* = Pulse, &c.

Potain noted that reduplication of the second sound occurred in health at the end of inspiration and beginning of expiration.

It may take place in conditions of debility of the muscular structure of the heart, and is often observed in the course of typhoid fever, commencing usually in the second week. In such cases M. Hayem has shown that there is an inflammation of heart muscle. The reduplication ceases as health is regained.

There is no doubt, however, that there is one condition in which the phenomena occurs most frequently and in its most pronounced degree—that is, in *obstruction at the mitral orifice.* Guttmann says it is heard in almost a third of the cases of mitral stenosis, and Dr. Hayden found it in thirty out of eighty-one. This accords with my own experience. Of twenty-seven instances of reduplicated second sound noted by Dr. Hayden, twenty-six were cases of mitral obstruction; you will conclude, therefore, that doubling of the second sound is a sign not only of physiological but of clinical and diagnostic importance; you will

consider it as presumptive evidence of *mitral stenosis.* Doubling of the second sound is also heard in cases of adherent pericardium (Friedreich) occasionally, and *passim* in engorgement of the right side of the heart, in fatty degeneration, and in some cases of pulmonary emphysema and of pulmonary tubercle; and accompanied by murmur, occasionally in disease at the aortic orifice. When you meet with it, note carefully the area over which it is heard, and determine which of its elements has the preponderance in given areas. Dr. Hayden says: "In every example of this anomaly which has come under my notice, the double character of the sound was exhibited only in the area over which the sounds of the aorta and the pulmonary artery were *both* audible; whereas when the stethoscope was shifted a short distance to the right or left of this region, a single second sound only was heard." * This does not accord with Guttmann, who says: "The broken diastolic sound is (so far as I have observed) certainly not loudest over the large vessels, but at the lower part of the sternum and near the apex of the heart."† My own observations accord with those of Guttmann.

There are few questions in cardiac pathology more difficult than that of the mode of causation of reduplication of the heart-sounds. This can scarcely be surprising when it is considered that observers are by no means agreed as to the causes of the normal, uncomplicated sounds of the heart. As regards reduplication of the *first sound*, the theory of which long seemed with greatest probability to be correct, ascribed it to non-synchronous contraction of the two ventricles. Thus, a right ventricular systole is followed by a left ventricular

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* "Diseases of Heart and Aorta," p. 165.

† "Handbook of Physical Diagnosis:" Sydenham Society's Translation, p. 278.

systole, or *vice versâ*. Many observers have, however, found it difficult to accept this theory on account of the structural unity of the ventricles, and the observed consentaneousness of their action. That such derangement of synchronism is possible is, however, some observers consider, well proved by physiological research.

Dr. J. Barr, of Liverpool, considers that each side of the heart has, to a certain extent, its own nerve-supply, and can accomplish its own peristaltic action, and, though both sides are set to the same time and have a complex interlacement of fibres, "it is an experimental fact that one side can begin or end contraction before the other."* In some rare cases, too, this want of synchronism on the part of the ventricles can be clinically demonstrated. Dr. Barr has described to me a case in which the impulse of the left ventricle could be first felt, and then that of the right ventricle, and this succeeded by a weak impulse of the left. On auscultation there were heard a loud systolic (mitral) murmur, then a sharp, clear first sound, followed by a short murmur (mitral). The proximate cause of the reduplication then, as advanced by those who first adopted the theory of asynchronous systole of the ventricles was the retarded closure of the tricuspid valve, owing to the excess of blood pressure in the right cavities. Dr. Barr, however, whose investigation of the subject is a very careful one, whilst adopting the theory of asynchronism of the ventricles, gives an opposite explanation of the *proximate* cause. He considers that the excess of blood pressure in the right heart (such as occurs in the normal heart during deep expiration, when physiological reduplication of the first sound is heard) stimulates the right ventricle to commence contraction before the left, so the tense closure of the tricuspid valves occurs before that of the mitral. One of the chief objections urged against the theory of ventricular asynchronism has been that the disturbance in systole ought in the nature of things to be accompanied by disturbance in diastole, that the non-simultaneous impletion of the aorta and pulmonary artery respectively should be followed by non-

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* *Vide* a Paper on Reduplication of the Heart-Sounds. *Medical Times and Gazette*, 1877.

simultaneous closure, by reflux of the aortic and the pulmonary semilunar valves ; that, therefore, doubling of the first sound should invariably be accompanied by doubling of the second, which is not the case. Dr. Barr meets this difficulty by advancing the view "that though the right ventricle commences its systole before the left, it is longer in emptying itself, owing to its overloading, and to the increased obstruction in the pulmonary circuit, hence, although it had the start of the left, it has not completed its contraction before it, so there is no reduplication of the second sound under these circumstances." Of other theories which have been advanced to explain reduplication of the first sound, may be mentioned that of Guttmann, which ascribes it to non-synchronous tension of the *individual segments* of the auriculo-ventricular valves owing to absence of perfect uniformity in the contraction of the papillary muscles. This hypothesis appears to me in the highest degree improbable; it would seem much more likely that such irregularities on the part of the papillary muscles could produce, not reduplications, but *murmurs*. Dr. Hayden's view is, that the doubling is equivalent to the resolution of the first sound into its two constituent elements—viz , the ventricular impulse and the click of valvular tension. According to this, the click of valve tension should occur at the very latest period of systole, the "thud" of muscular impulse always preceding it. This cannot, however, be sustained, for cardiographic evidence abundantly shows that the closure of the auriculo-ventricular valves occurs early in the systole, and that the first sound is prolonged subsequently to their closure. The "click" therefore ought always to precede the "thud," whereas Dr. Hayden says that, in his experience, without exception, "the first element of the double sound has been dull and muffled, and the second sharp and clear."*

Dr. George Johnson has advanced a theory totally distinct from the others. He considers that the so-called reduplicated first sound consists of really the rapidly following sounds of first an auricular and then a ventricular, systole. *The contraction of a dilated, and especially of a hypertrophied auricle becomes audible, and the first division of the double first sound is the result*

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* "Diseases of Heart and Aorta," p. 118.

of the auricular systole. That the sound can be the *direct* effect of the systole of the auricle is very difficult to believe. As well as other observers I have had opportunities of auscultating the auricle in very many conditions of dilatation and hypertrophy, and when it has been through pulmonary disease totally uncovered by lung, but in no case has there been a particle of evidence that its muscular contraction is accompanied by sound. Nor does it seem probable, when we consider how feeble is the muscular sound of the ventricle, even in conditions of the greatest hypertrophy. Whilst denying that the sound is the direct effect of the auricular systole, I would by no means assert that it may not be the indirect effect thereof. It is quite conceivable that in some cases the energetic systole of the auricle suddenly floating up the curtains of the mitral valve, may so put them on the stretch as to cause a sound—the click of valve-tension. Potain noticed a form of seeming reduplication of the first sound, which he termed "*bruit de galop*" in cases of hypertrophy of the heart associated with granular kidneys. He considered that the first element of the apparent reduplication was presystolic. Guttmann also has observed such presystolic sound in a number of cases of cardiac hypertrophy; he considers that the auriculo-ventricular valves are to a certain extent rendered tense at the end of diastole—that is in the presystole; but this tension is so feeble that no sound results; when, however, from hypertrophy of the auricle the tension is increased, it is quite possible that sound may be occasioned.

As I have said, reduplication of the first sound is a phenomenon of comparative rarity. Many good observers have never met with a case. Dr. Hayden has noted twelve examples—eight in cases of females, four in males. Of these, six were considered nervous; three associated with menstrual disturbance; one with gout; another with Graves' disease. Hypertrophy of the left ventricle existed in three cases; dilatation (in connection with cirrhosis of the liver) in one case; and fatty degeneration in two cases. Mitral stenosis existed in one case; in this both sounds were double.* Dr. Hayden says: "Reduplication of the first sound is so intimately associated with simple or eccentric

* "Diseases of Heart and Aorta," p. 164.

hypertrophy that, if carefully sought, it will be found in every such case anterior to consecutive degeneration of tissue." I have already said that I can by no means accept Dr. Hayden's theory of a splitting-up of the normal first-sound into its two component elements of muscular susurrus and valve tension. It appears to me that either (*a*) the reduplication is real and caused by non-synchronous tension of the auriculo-ventricular valves of the right and left sides, or (*b*) it is only apparent and due to a presystolic being closely followed by a normal first sound, the presystolic sound being produced by the sudden floating upwards of the mitral curtains, occasioned by the auricular systole. I have met with reduplication, or apparent reduplication of the first sound in eleven cases. Of these the conditions were various, but classification could, in my opinion, be made as follows:—(*a*) cases (four) in which there was evidence of prolonged ventricular systole, with rough first sound or murmur (in one case double murmur) at the base of the heart, and a pulse of decidedly high tension; (*b*) cases (three) in which there was a disturbance of respiration and evidence of, at least temporary, distension of the right chambers of the heart; (*c*) cases of mitral stenosis (three); (*d*) one case of myocarditis in course of typhoid fever; here both first and second sounds were doubled. The positions of audibility of the reduplication were over the apex, five cases; over the base, two cases; generally over the præcordium, one case; over the right cavities, one case; and in two cases its audibility varied from apex to base. In *a*, *b*, and *d*, it would seem quite probable that the theory of asynchronism of the pulmonary and aortic second sounds might be justified; but in group *c*, it appears to me that there are strong probabilities that the apparent reduplication is really a presystolic succeeded by a systolic sound. This view may be best illustrated by the following brief abstract of a case:—Female, aged thirty, admitted under my care at the London Hospital, for epilepsy. Subacute rheumatism occasionally from age of six. Pregnant. The physical signs as regards the heart showed briefly the apex in normal position; first sound subdued but accompanied by murmur conveyed towards axilla. At base, second sound pronounced, but no other abnormality. On the day following admission, I found that the first sound was reduplicated, the reduplication was heard in a line leading from the fourth

interspace to the anterior border of the axilla. Two days afterwards the patient aborted. The reduplication persisted but changed in certain characters, at the end of a fortnight it was heard from the third interspace to the apex; a little *internal* to the apex reduplication was distinct and sounds uncomplicated. On nearing the apex a soft systolic murmur tailed off from the *second* element of the reduplication, and at the apex, reduplication was lost, a first-sound murmur only being heard. Sphygmographic tracings gave evidence of hypertrophy of the left ventricle; there was an ample tidal wave and the dicrotic was high in the tracing. The trace also showed occasional disturbance of rhythm (double pulse), and the diastolic periods were unequal in time. Cardiographic evidence confirmed this diagnosis of hypertrophy. In some tracings there was evidence of the point of closure of the auriculo-ventricular valves: the summit of the systolic portion was broad, and the terminal swelling marked. In the diastolic portion, the auricular eminence was chiefly remarkable for the breadth of its base. This case tells in favour of the view that the second element of the reduplication was the voice of the left ventricle. This is shown by the fact that the murmur which started from it was mitral systolic. To what then was due the first element? It seems scarcely likely that it could be the voice of the right ventricle—of the tricuspid valve—for reduplication was at first not even audible over the right ventricle. It would seem to me more probable that it was due to the presystolic flap of the mitral valve in the manner I have before mentioned. This view was, I think, confirmed by the progress of the case: for, after losing sight of the patient during the interval, I had an opportunity of examining her again eight months subsequently, *then* there was no reduplication of the first sound but *a presystolic murmur, typical and distinct*. I am convinced that in certain cases of mitral stenosis a phenomenon closely or exactly simulating reduplication of the first sound may be observed. (See Part II.) In one case of mitral stenosis, in which I noted this close resemblance to reduplication of the first sound, the cardiographic tracing showed a very broad, pronounced eminence, indicating the auricular systole; the inference was plain that there was much hypertrophy of the auricle and the degree of stenosis slight, so that the auricular contraction gave a distinct impulse to the

apex. The mechanism of production of such sound seems to me by no means difficult to realize. During the interval immediately succeeding the relaxation of the ventricle, the blood, subject to the tension in the left auricle and pulmonary veins has been pouring into the ventricular cavity; the fluid naturally finds its way in the direction of least resistance, that is, its course, when impelled towards the apex, is round the walls of the ventricle, thus coming *behind* the curtains of the mitral valve, and bellying them out (so to speak) as the sails of a ship are bulged by the force of the wind. At the moment of auricular systole, the ventricle, as yet only partially full, is rapidly distended, the force of contraction of the auricle giving an impulse to the apex of the ventricle, and, as may of course be inferred, giving a *contre-coup* to the already partially-strained mitral curtains. In normal conditions such *contre-coup* is inaudible, but when the auricle is more than ordinarily powerful, or when the mitral valve is so changed as to give rise to the sound of membranous tension, it becomes perceptible closely preceding the sound produced by the ventricular systole—that is, the sound of complete closure of the valves guarding both auriculo-ventricular orifices plus the muscular sounds of the ventricles. The sharp, short, first sound so common in mitral stenosis I believe to be chiefly due to sudden tension of the *tricuspid* valve. In this I know other observers, especially Dr. Barr, agree with me. I would by no means urge, however, that such must be the universal interpretation of cases of reduplication of the first sound. Infrequent as are the instances, their very incongruity teaches that each case must be judged for itself. For cases of physiological reduplication, Dr. Barr's theory offers, to my mind, a plausible explanation. So also it may explain others connected with neurotic disturbance. I find it difficult, however, to associate reduplicate first sound in any causal way with differences of intra-ventricular pressure, seeing that long-persisting variations in such pressure and disturbances of balance must be so common, whilst the phenomenon itself is so rare. As regards its probable auricular origin, in some cases we may get more evidence in our consideration of reduplication of the second sound. A reference to A. B. Fig. 3 will show the mutual relations of these reduplications. Now supposing it were granted that the sound-producing tension of the mitral

curtains might be effected in some cases early in diastole, and not only always late in diastole—that is, in presystole—we

FIG. 3.

Diagram showing positions in the cardiac rhythm of *apparent* reduplications of A the second, B the first sound.

might have in some instances a semblance of reduplication of the first, in others of the second, sound. If the tension happened to be effected late, or in presystole, it would be represented by B, in the diagram—apparent reduplication of the *first* sound; if early, by A, apparent reduplication of the *second* sound.

So-called reduplication of the second sound is a phenomenon which observers agree to be much more frequent than that we have just considered. It is undoubted that by far the most common condition in which double-second sound is heard is mitral stenosis. Next in point of frequency, according to Dr. Hayden, is aortic insufficiency. With considerable unanimity the phenomenon has been ascribed to *a want of synchronism in the closure of the valves of the aorta, and of the pulmonary artery respectively.* The sequence of closure may vary. In cases of aortic insufficiency, Dr. Hayden is satisfied that the second element is aortic, because the murmur of aortic regurgitation accompanies it. He believes that in such cases the left ventricle is dilated, and so is slower to become emptied in systole; consequently the aortic reflux is postponed. On the other hand, in mitral stenosis the derangement is chiefly in the pulmonary artery, "the entire pulmonary system and the right chambers being engorged by obstruction at the mitral orifice. In the effort to overcome this obstruction, the systole of the right ventricle is protracted, and the reaction of the pulmonary artery proportion-

ately postponed. The reaction of the aorta is, on the other hand, in mitral stenosis most probably anticipated, where the left ventricle is reduced in capacity, as always is the case where mitral or aortic reflux does not co-exist. Hence it is likely that in simple mitral stenosis two causes of doubling of the second sound are in operation—viz., diminished capacity of the left, and dilatation of the right, ventricle" ("Diseases of Heart and Aorta," p. 128). It seems a truism to assert that conditions of blood-pressure in the two great vessels respectively, cannot be the *vera causa* of such reduplication; for until the occurrence of diastole, there can be no reflux, and consequently no second sound. The causation of reduplication, as of the first so of the second sound under the theory we are now discussing, *must* revert to the ventricles. When their systole is simultaneous, the second sound, produced by reflux against the semilunar valves of the two great vessels, at the moment of their relaxation, must be simultaneous also. When their systole is not simultaneous, or does not last through equal periods, the diastolic reflux cannot be simultaneous; if the discrepancy be such as can be appreciated by the ear, the second sound is doubled. The following is Dr. Barr's view:—"As I have ascribed reduplication of the first sound to asynchronism in the initial stage of ventricular contraction, so I believe reduplication of the second sound to be due to asynchronism at the end of contraction, and in the consecutive reaction of the aorta and pulmonary artery, with the tension of their respective valves." The comparative rarity of cases of reduplication of the first sound is explained, by Dr. Barr, by the much greater length of time occupied by the first than by the second sound, whence is required a correspondingly greater want of synchronism between the closure of the tricuspid and mitral valves than between the pulmonic and aortic, to produce a reduplication. *Physiological* reduplication at the end of inspiration is accounted for by the fact that the capacity of the pulmonic system being increased, the obstructive burden upon the right ventricle is lessened, consequently the systole of the latter is shortened. The lessening of the duration of the systole of the right ventricle implies anticipation of the diastolic closure of the pulmonary semilunar valves—so there is a reduplicate second sound, the primary element being pulmonic. In

pathological reduplication, the origin may be in either the right or the left ventricle. If from any cause the systole of one of the ventricles be protracted, the diastolic closure of the valves of its great vessel of exit will be delayed also, and so the second sound will be double. Guttmann supports the view of non-simultaneous closure of the aortic and pulmonary valves, without giving reasons for the proximate cause. He says, however, that it is not unreasonable to explain the phenomenon by a change in a single set of the semilunar valves, causing their tension to take place in two distinct moments. This hypothesis appears to me impossible to accept; such change would surely be likely to produce murmur rather than reduplication. But Guttmann says that reduplication of the second sound in mitral stenosis is difficult to account for satisfactorily, and in this I quite agree with him. He adds: "The broken diastolic sound is (so far as I have observed) certainly not loudest over the large vessels, but at the lower part of the sternum, and near the apex of the heart, and is further absent in those more marked cases of mitral contraction, precisely in the cases in which the conditions most favourable to the postponement of the closure of the pulmonary valve are present in their highest degree." Reduplication, moreover, does not occur in mitral regurgitation where the conditions of relative blood-pressure are profoundly altered. Guttmann considers it probable that the reduplication may arise at the narrowed mitral orifice itself; that it may be a component part of a presystolic (or, as he terms it, diastolic) murmur, and adds that it has been conjectured that the first element of the reduplication is the diastolic pulmonary sound, and the second is produced towards the end of diastole by the contraction of the hypertrophied left auricle ("Physical Diagnosis," p. 279). In fact, the hypothesis reverts to the last I have mentioned as probably explaining some cases of reduplication of the first sound. I consider then that there is a theory of auricular causation of reduplication of the second sound which is well worth considering and that we have before us two plausible explanations of the phenomenon:—

A. That it is due to non-simultaneous closure of the semilunar valves in the aorta and the pulmonary artery respectively.

B. That it is the effect of a sudden tension of the mitral curtains after the normal second sound.

I now proceed to discuss the evidence on this question, which my own cases have afforded me. As I have said, I have observed reduplication of the second to be far more frequent than that of the first sound, and in a vastly preponderating number of cases the phenomenon is associated with *mitral stenosis.* It has occurred in about a third of the cases of mitral stenosis which I have examined;* it would therefore appear *à priori* probable that in this form of valve-tension there should be some special reason for its occurrence. I will consider first the evidence of my *cases of reduplication of second sound in conjunction with mitral stenosis.*

The following case is illustrative:—A lady of fifty was frequently under my care for symptoms of dyspepsia; there was no history of rheumatism nor development at any time of articular phenomena. I had frequent opportunities of examining the heart, but found no lesion. After a few months, however, during which no special symptoms had manifested themselves, the patient complained of fluttering of the heart, and I then found a rough presystolic murmur just internal to the apex, with a short systolic murmur heard at and outside the apex. There was *distinct reduplication of the second sound* heard at the aortic cartilage and down the left border of the sternum, but ceasing at the point internal to the apex, where the presystolic murmur became audible. At the next examination the reduplication was heard as far as the apex. A subsequent note states, "reduplication very marked, but no presystolic murmur heard." Afterwards, "no reduplication heard at any part of base; commences well below the region of the pulmonary valves, and is certainly loudest at or even a little outside the apex." A subsequent note: "Reduplication still very pronounced; not heard at left base nor anywhere left of the sternum until the level of the fourth costal cartilage; it is well heard at the apex, and marked in the axilla." Afterwards it was audible at the left scapula.

We will ask now whether these phenomena can be explained by the theory which postulates want of synchronism between the aortic and the pulmonary second sounds. Supposing this

* Strictly speaking, eleven cases in 37.

theory to be correct, it would be the *aortic* that would be the first element of the reduplication; because, as the aortic region was receded from, the second element of the reduplication undoubtedly became more and more pronounced. But to accept this theory even under the conditions first noted, we must agree that the aortic second sound was very loud, not only over its normal area, but as far as the apex. We had in this case, however, abundant reason for knowing that the aortic tension instead of being above was far below the normal. A reference to the record of one of Dr. Hayden's cases points to the same difficulty. Here "the second sound was double, and heard all over the præcordium." Under the accepted theory, therefore, the aortic second sound, its first element, must of course have been audible all over the præcordium, that is, over a wider area than often the normal aortic second sound is heard. But the case was well observed and diagnosis confirmed by post-mortem examination, and Dr. Hayden adds: "The quantity of blood, which in this case, passed into the aorta at each systole of the left ventricle must have been very small, owing to the twofold lesion of mitral obstruction and incompetency." Moreover, the aortic orifice was reduced to the diameter of the point of the little finger, and its valves thick, and therefore less disposed to give the "click" of closure. The difficulty of accepting the asynchronism theory is further greatly increased by consideration of the subsequent areas of audibility, for the reduplication was not even *audible in the aortic area*. On the other hand its area of audibility became nearer to the apex, and afterwards included, not only the apex, but the region of the axilla external to it and even the back. Seeing, therefore, that the reduplication was manifest in areas in which even the normal aortic second sound is often inaudible, I conclude that, in this case, the latter could have had no share in producing the phenomenon of reduplication. In not more than half the cases of reduplicated second sound in mitral stenosis which I have observed, has such reduplication been audible in the aortic region. It is almost invariably heard best down the left border of the sternum up to the point at which the presystolic murmur becomes manifest.

It seems to me, therefore, that reduplication of the second sound in cases of mitral stenosis can be best explained thus:

The first element of the reduplication is the normal second sound; the tension in the aorta being feeble, it is the *pulmonic* element which has the chief share in the production of such second sound. The second element of the reduplication is the sudden tension of the abnormal mitral-curtains produced after the relaxation of the left ventricle. I am not prepared to say that systole of the auricle is *essential* to produce this sudden tension; it may be quite possible that the reaction of the distended pulmonary veins and left auricle may be sufficient to cause it. So it may occur previously to the auricular contraction, the latter occasioning or reinforcing the presystolic murmur, separated by a slight interval from the second element of the reduplication.

I will now consider the evidence in the cases of reduplication of the second sound, which I have met with *not* in association with mitral stenosis. Of such there were eleven examples, which I consider could be ranged under two categories. I. Cases in which there were troubles of respiration (broncho-pneumonia, phthisis, dilatation and signs of failure of the left ventricle, and especially dilatation of the *right* chambers of the heart). In this class were eight cases. It is noteworthy that in all in which the area of audibility of the reduplication was noted, this was either that of the *origin of the pulmonary artery* or else of the apex of the right ventricle. In this respect, therefore, the cases were strikingly dissimilar from those in which the reduplication was associated with mitral stenosis. I am quite disposed to think that the theory of asynchronism of the two second sounds would best explain them, and that Dr. James Barr's view as applied to explain the physiological doubling of the second sound—*i.e.*, the assumption that the systole of the right ventricle is *shortened*—is in these particular pathological conditions probably correct. The other class in which I have found doubling of the second sound in the absence of mitral stenosis is:—II. Cases in which there was hypertrophy of the left ventricle, with high tension in the arteries. In these it is to be noted that the area of audibility of the reduplication was more variable, more extensive, and especially approached the apex more closely than in those of the former group. In the case of a female, aged fifty, with chronic renal disease and hypertrophy of the left ventricle, the reduplication, as observed at different periods

was, (1) the heart's apex; (2) base as well as apex; (3) base only and maximum in aortic area. Post-mortem examination showed great hypertrophy and dilatation of left and right chambers. In the three cases in this category it would be quite legitimate to infer that there was protracted systole of the left ventricle, and that the right ventricle having more quickly accomplished its contraction, reflux against the pulmonary valves took place at an earlier period in diastole than reflux against the aortic. Such would accord with Dr. Barr's theory, and a case which I had an opportunity of closely observing, convinced me that *if there were such asynchronism*, the pulmonary second sound was the *first* element of the double sound. Whilst the probabilities are here also in favour of the theory of asynchronism, I think it not impossible that the phenomena *may* be explained by my theory of diastolic tension of the mitral curtains as in the cases of mitral stenosis. On this point we require more evidence. As practical points I would urge that in any case which seems to present reduplication of the sounds of the heart, you should endeavour with care to establish whether such reduplication is real or apparent. Remember that reduplication of the first sound may be simulated by a presystolic sound occasioned (as I have described) by the systole of the left auricle. In apparent reduplication of the second sound, remember that there is *presumptive* evidence of mitral obstruction; in a minority there is a disturbance of rhythm owing to comparative feebleness of the right ventricle, or to hypertrophy with slow contraction of the left.

You will meet with many instances of disturbed rhythm of the heart which are exceedingly puzzling, but by attention and repeated examinations you will be able to resolve them in most cases. A tumultuous and irregular succession of sounds, which at first seem chaotic, may frequently be analysed, and found due to a definite association of morbid and normal heart-sounds.

LECTURE VI.

AUSCULTATION.

PART II.

Abnormal sounds—Pericardial friction-sound—Relations between pericarditis, rheumatism and renal disease—Influence of posture on friction-sound—Exocardial friction—First sound murmur over aortic valves—Anæmic murmurs—Murmurs in chorea—Aortic stenosis—Ulceration of aortic valve-segments—Aortic second sound murmur—Evidence from ophthalmoscopic examination—Aortic insufficiency—Physical cause of murmurs—Conduction and convection of murmurs—Double aortic murmur.

HAVING considered the modifications of the normal sounds, we will now turn to our second subdivision and assume :—

(*b.*) THAT ABNORMAL SOUNDS ARE HEARD OVER THE HEART REGION.

Abnormal sounds, heard at, or close to, the situation of the heart, acompanying the cardiac movements and apparently mingling with some of the normal sounds, may have their origin (1) in the structures external to the heart and pericardium ; (2) in the pericardial sac ; (3) in the muscular tissues of the heart ; (4) in the endocardium and the valves of the heart ; (5) in the blood transmitted by the heart.

As regards the first-mentioned cause of abnormal sound, it is of course assumed that you have carefully estimated the pulmonary conditions, especially of the

left cavity of the thorax and the lung which borders on the heart region. The diseases which you have chiefly to consider as inducing sounds which resemble those due to intrinsic diseases of the heart, or as modifying and irregularly conducting the sounds due to intrinsic diseases, are pleurisy with viscid effusion, cavities in the pulmonary substance near the heart, and condensation of the lung. The differential diagnosis of these conditions we shall consider as occasion arises.

We will assume that:

(1.) A rubbing or creaking sound, accompanying both movements of the heart, is heard superficially over the cardiac region, but is not intensified at apex or base.

This indicates a diseased condition of the pericardium in which the visceral and parietal layers are separated by a viscid or fibrinous effusion, or in which the pericardium itself has become thickened or roughened. Your previous examination may have given a clue to your diagnosis. You may have concluded that the pericardial sac is more or less distended, and the symptoms may have assured you that there is in existence a pyrexial disease accompanied by cardiac, pulmonary, and, perhaps, cerebral phenomena. You may even have had the more positive evidence of pericardial friction fremitus. Moreover, you may have heard the friction-sound by direct application of your ear to the chest before commencing to use your stethoscope. If you suspect pericarditis you should, in my opinion, never omit the method of direct auscultation.

But you may have none of these signs to guide you, and let me insist strongly on this. Your patient may have pain referred to some of the joints; these pains

may be very trivial in character, may, indeed, be absent; pyrexia may be very slight or inappreciable. I mentioned in an early Lecture a case of pericarditis in the most pronounced degree which went through its course without a single symptom of distress. I have lately had under my care, at the North-Eastern Hospital for Children, a boy who manifested the disease with its grave progressive course and complications, but with the occurrence of so few subjective symptoms that it was impossible for me to persuade the parents to leave the child as an in-patient, and he was brought to me bi-weekly as an out-patient. In this case there was loud pericardial friction, which, as it faded away, gave place to the murmur of mitral regurgitation, indicating that endocarditis had attacked and spoiled the mitral valve; at the next consultation it was evident that the disease had spread to the aortic valves, for there was a murmur of aortic obstruction, and at the following there was, in addition, a murmur of aortic regurgitation, showing that the endocarditis had rapidly destroyed the integrity of the aortic valves and induced their most dangerous lesion. You will remember, then, that you are not to look for pronounced symptoms to suggest to you the occurrence of pericardial friction.

I have said that your patient may complain of pain in one or more of the joints. This rule you are most strongly to observe—carefully to examine the heart and to suspect pericarditis in any case manifesting acute or subacute rheumatism. By far the most common form of pericarditis is that which is associated with rheumatism. Many circumstances govern the frequency of the manifestation of pericarditis in rheumatic fever, and authors vary in their estimate of such

liability even from 16 to 37 per cent. I have compared the figures recorded by Fuller, Von Bamberger, Roth, Leudet, Duchek, and Chambers, and I find that they give about 24 per cent. as the ratio of cases of pericarditis occurring in acute rheumatism.* Of 50 cases of recent pericarditis, Flint observed that 19 were manifestly rheumatic. Even this high figure, however, does not represent the prevalence of the rheumatic form of pericarditis; for I have already drawn your attention to the fact that pericarditis occurs in the subacute form of rheumatism, and in cases where the articular phenomena are very slightly developed. In some rare cases the process of rheumatic fever commences by pericarditis.

Another condition of disease in which you must recollect that pericarditis is probable, is renal disease. In all cases of pericarditis, but especially in cases occurring after adult age, you should examine the urine for albumen. This is highly important from the point of view of prognosis, for whilst rheumatic pericarditis generally ends in recovery, pericarditis occurring with renal disease is generally fatal. The disease may also develop in morbid conditions of the blood as scurvy, and, though rarely, in the course of scarlatina or variola.

Your previous examination will, in most cases, have indicated that the pericardium contains some liquid effusion; but this is not invariable. Pericarditis, with friction-sound, may occur, when the exudation is inappreciable (pericarditis sicca), or you may examine

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* The results of the following observers approach very closely and are probably nearest the truth: Fuller, 16·7; Duchek, 16: Chambers, 18 per cent.

the case at a period when most of the effusion has disappeared by absorption.

The importance of the observation of the sound of pericardial friction is very great. This is one of the very few great points that we have gained since the time of Laennec. In most cases it is marvellous to see how close and complete were the observations of this great master. Our advances in physical diagnosis since his day, until lately, have been neither great nor rapid; but in this case there is an exception. Laennec says:—"There are few diseases more difficult to recognize than pericarditis, or more variable in their symptoms. . . . I must acknowledge that mediate auscultation does not afford much more certain signs of pericarditis than the study of the general and local symptoms." Thanks to the subsequent observations of Stokes, Watson, and Bouillaud, we now know that the existence and course of pericarditis can be traced with precision as in the case of other cardiac diseases.

It is very rare indeed that a friction murmur fails to be developed at some time during the evolution of the pericarditis. It appears early in the disease. Sometimes it disappears also early. Walshe has known it appear and disappear within six hours. Sometimes it appears, then disappears for a time during a period of greater abundance of liquid effusion, then reappears, to vanish gradually. Most frequently it lasts for many days, varying in character and intensity, and is heard over a less and less extended area as the disease terminates.

The characteristics of the pericardial friction-sound are chiefly—(1) Its quality; it is a "rubbing" or "creaking" sound, resembling that produced by the attrition of two surfaces of cloth or leather. It accom-

panies both movements of the ventricles, and is admirably indicated by the expression of Sir Thomas Watson—a "to-and-fro" sound. This rule is subject to very rare exception when the rub is heard only with the systole. We must postpone consideration of this till we come to the differential diagnosis of valvular murmurs.

It is by no means uncommon to find that the rub of pericarditis has a *triple* rhythm. This was shown by the late Dr. Hyde Salter to be due to the fact that roughening occurred not only over the ventricles but over the auricles. The sound over the auricles is presystolic, then follows the friction-sound produced by the systole of the ventricles, and the third portion is produced by the diastole of the ventricles.

(2) Its limitation. It is heard only over the superficial cardiac region. This is very important. The sound is not conveyed in certain directions from the heart-area, as we shall find to be the case in regard to endocardial murmurs. It is situated over the heart-muscle, and is associated more with the *movements* than with the normal *sounds* of the heart. At apex and base these normal sounds may be heard—distant and feeble if there be much effusion. On applying the stethoscope over the intervening area occupied by the heart-muscle (chiefly the right ventricle), the rubbing sound becomes manifest. Pericardial friction appears earliest, and most frequently, at the *base* of the heart, near the great vessels. (3) Its superficiality. It seems to be generated near the ear. If you withdraw your ear slightly from the chest, you may still hear the sound. Dr. King Chambers calls attention to the following plan, as affording valuable evidence:—Having observed the sound with the ear, as usual, close to the stethoscope, gradually withdraw your ear, the stethoscope remaining applied to the chest, the sound of pericardial

friction will still, with great probability, be recognizable. This nearness of sound to the surface is valuable in the differential diagnosis between exocardial and endocardial murmurs.

The friction-sound, however, may be very slight and faint. In such case you should make your patient change posture; the friction may then become more manifest. The intensity may be increased by a change from the vertical to the recumbent position, or *vice versâ*, or by inclining the body backwards or forwards. In some cases a friction murmur is only heard when a particular position is assumed by the patient. When, therefore, your examination has led you to suspect pericarditis, especially when you are satisfied that there is effusion into the pericardial sac, and you are anxious to discover evidence that the exudation is fibrinous and tending to absorption, you should carefully auscultate in various positions, and note the presence or absence of friction-sound.

Attention to the points I have mentioned will generally enable you to recognize the existence of a pericardial friction-sound; but there is one condition in which the diagnosis may be difficult. This is when the friction is produced, not within the pericardial sac, but outside, in the pleura.

You will remember the rule I adduced—to examine carefully for disease in the left side of the chest. Suppose that your examination has led you to conclude that there is pleurisy with effusion, or pneumonia, or that the lung abutting on the heart-region has undergone any of the changes occurring in the course of pulmonary phthisis. If, in such conditions, you hear a "to-and-fro" sound over the præcordium, remember that this may be a *pleural* friction, modified by the

movements of the neighbouring heart, and so made to resemble the pericardial rub. The pleural surface may be locally roughened by recent exudation, or thickened by more remote, or by progressive inflammatory changes. If you have such a sound under such conditions, and there is no other evidence of the existence of pericarditis, the balance of probability will be in favour of its exocardial origin. To further the differential diagnosis, observe whether the sound varies in character, and what are its relations with the rhythm of respiration. Does it alter in intensity in an irregular manner? Does it become imperceptible in some of the cardiac revolutions? Is it more pronounced at the end of a full inspiration? Observation of these points will help you to a correct conclusion.

A pericardial friction-sound is less liable to variation than a pleuro-pericardial: the latter is most evident when the lung is inflated, and thus the rough pleural surface more closely adapted to the pericardium, whilst the pericardial sound is most pronounced when the heart is least covered by lung—that is to say, at the end of expiration.

Having excluded the sounds which take their origin in the structures superficial to the heart, we come now to a wide field of inquiry—that which includes the morbid sounds intrinsic to a diseased heart. The subdivision of abnormal sounds which we now consider is that wherein:

(2.) Abnormal sounds, occurring with or replacing the normal sounds are heard only, or with a maximum intensity, over the various situations of the valves, or in definite relation with such situations.

You have applied your stethoscope over the aortic cartilage (the point where the second right costal

cartilage joins the sternum), and you hear a soft blowing sound; placing the tip of your finger over the apex of the heart, or over the carotid artery, you are convinced that the murmur coincides in time with the pulse—that is to say, with the first sound of the heart. Moving your stethoscope so as to auscultate in a direction upwards beneath the right clavicle or downwards over the heart, you find that the sound is lost. It is, in fact, *a first sound murmur localized over the situation of the aortic valves*, and it indicates an alteration of the segments of these valves or a change in the normal aortic orifice: the form and nature of such change we shall consider presently.

Suppose, however, that you are satisfied that *whilst the sound which I have just mentioned is not propagated downwards towards the heart, it is conveyed in a line extending towards the right clavicle.* This may possibly be due to an *aneurism* of the ascending part of the arch of the aorta, or of the innominate artery. To establish or eliminate this hypothesis, you must examine carefully for the concurrent signs of such condition—a local prominence, pulsatory to the touch, and perhaps communicating a thrill, a localized area of dulness over the affected vessel, signs of alteration of circulation in the distal branches of the diseased vessel, and effects of the progressive pressure on neighbouring structures, caused by the growth of the aneurismal tumour.

You must remember, however, that whilst the occurrence of aneurism may explain the existence of the murmur, the hearing of this murmur is by no means necessary to establish the existence of aneurism. A systolic murmur is absent in a large number of cases of aneurism.

Aneurism eliminated, you have next to consider whether the murmur may be *anæmic*. Here let me say that you may find the differential diagnosis not at all easy. Carrying your stethoscope in the direction of the right subclavian artery, the right carotid, the left subclavian and the left carotid, note the points where the murmur disappears, and, if this happens, where it reappears. In the great majority of cases an anæmic murmur is a soft murmur: it is heard in the course of the great arterial vessels, not with a diminishing intensity, as one recedes from the heart, but often with reinforcement over the arteries: a slight increase of pressure made by the stethoscope increases the loudness of the murmur or develops it when it is not heard. There is one observation which will give you positive aid in the differentiation. Having caused your patient to turn the head towards the left, apply your stethoscope above the right clavicle in the hollow behind the sterno-cleido-mastoid muscle: you may now hear a continuous musical hum, the origin of which is in the great veins. You can at once distinguish it from the murmur which you have just heard in the arteries, because, whilst the latter is systolic, occurring only with the arterial pulse, the former is an uninterrupted sound, a sound called by French observers the *bruit de diable*, "humming-top sound." By making pressure with the finger over the veins above the stethoscope, the murmur will be made to cease—a sufficient proof of its venous origin. You should auscultate in like manner on the left side of the neck. If you hear the venous hum in either or both of these situations, you have strong evidence that the murmur which you have heard at the base and up the vessels is a so-called

hæmic or anæmic murmur. But you obtain very valuable collateral evidence from the general condition of your patient. If there are the pallor and usual signs of anæmia; if your patient is a female of an age when the catamenia are commencing, or of a later age, when there are troubles due to excess or deficiency of the menstrual function or leucorrhœa, or of a still later age, when there has been much loss of blood—or if the case is one of a male, who has suffered from hæmorrhage or some potent debilitating cause, or presents the signs of early phthisis, the probability of the murmur being anæmic is rendered very great.* I have seen it stated that anæmic murmurs are seldom or never met with in young children. I can only say, that at the North-Eastern Hospital for Children, where we do not admit patients above the age of twelve years, I have had many opportunities of demonstrating this form of murmur.

"Cardiac murmurs of hæmic origin are invariably basic, they are loudest at midsternum or in the anatomical site of the orifices of the aorta and pulmonary artery. With this point as centre they have a diffusion area of three to four

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* I find, from notes taken by myself, of 57 cases under my own care where I discovered uncomplicated anæmic murmurs, that 15 only were males. The ages were, under sixteen, 7 cases; from sixteen to twenty-four, 18 cases; from twenty-four to thirty-two, 15 cases; from thirty-two to forty, 6 cases; from forty to sixty-six, 4 cases. The arterial anæmic bruit was heard at the base as well as in the carotids and subclavians of both sides, in 8 cases; in both carotids in 11 cases; in left carotid and subclavian, in 6 cases; in right carotid and subclavian, in 4 cases; in right carotid only, 1 case; in both subclavians, 2 cases; in left subclavian, 6 cases; at limited area over base not propagated up vessels, 16 cases.

inches in diameter, according to the intensity of the murmur and the conducting qualities of the chest-wall; but they never exhibit a definite line of propagation, as is the case with organic murmurs in this situation."—Hayden. "Diseases of Heart and Aorta," p. 252.

These murmurs, according to Guttmann, occur most frequently at the pulmonary orifice, and very seldom at the aortic.

I have said that you may find difficulties in determining whether a murmur be inorganic—*i.e.*, hæmic, due to the causes just considered; or organic—*i.e.*, due to structural change of the aortic valves. This difficulty occurs especially when the murmur is localized at the aortic cartilage, and when, as is sometimes the case, a general condition of anæmia develops or reinforces a murmur, due to structural disease. We shall lessen the difficulty when we have contrasted the organic with the inorganic basic murmur.

In the case of the soft murmur, which we are still considering (for we shall hereafter notice the loud basic murmur), the difficulties in diagnosing between the anæmic and the structural may be great.

I have occasionally found murmurs in anæmic subjects closely to resemble a *double aortic*—*i.e.*, systolic and diastolic. In such cases you will find that the systolic is the hæmic souffle heard at the aortic base, and generated either in the aorta, the subclavian artery, or in the pulmonary artery; and the seeming diastolic is really a continuous venous hum generated in the great veins of the neck. This continuous hum is overpowered during the systolic period by the arterial murmur, but it is audible during the diastole:—so the murmur appears to be to and fro.

The cases wherein the chances are in favour of its being structural will be, as I consider, in two classes—one in young subjects, where there has been a his-

tory of rheumatism, and especially where there is, or has been, chorea; the other in patients past middle life, where there is a probability of atheroma. In these cases there may be but slight impediment to the onward course of the blood in the aorta. It is very seldom that the aortic valve is much affected by the ordinary form of rheumatic endocarditis without the mitral valve being affected first or coincidentally. Bear in mind that we are now considering a soft murmur, heard alone over the aortic valves, without discoverable alteration of the other valves. There is a condition of the aortic valve in which the edges of its segments are fringed by little villosities, so-called vegetations; these may give rise to a very soft aortic murmur with the first sound. This condition is not necessarily an accompaniment of rheumatism, but where a valve is diseased by rheumatism or by atheroma, it is more apt to occur. In chorea, as we shall consider hereafter, the vegetations are more frequently found fringing the *mitral* valve, but they do occur on the aortic segments. In patients after middle life, such vegetations may be detached from the valve by the force of the current of blood, and being carried into the arteries, may plug one of the arterial branches. Such may occur in various parts of the system, but especially in the brain. Occurring in the small terminal arteries of certain parts of the brain, it is considered with great probability to be often the cause of chorea; when blocking a larger trunk, it causes various forms of paralysis, or attacks which are ascribed to apoplexy. Remember, therefore, the possibility of arterial embolism when you hear a soft murmur localized at the aortic cartilage in cases at the predisposing ages I have just alluded

to, and wherein you do not find anæmia to be a direct explanation of the phenomenon.

So also you may turn the argument round and conclude that when, with these predispositions and under these circumstances you hear a soft first sound murmur localized over the aortic cartilage, such murmur is probably due to a slight obstruction of the aortic orifice, and that vegetations may fringe the valves.

We will now suppose that instead of the soft murmur we have been considering, there is a *loud bruit heard over a wide area, but having its maximum intensity over the aortic cartilage*. When you commence to auscultate the heart region, you are at once cognizant of a loud rough systolic murmur; over the second right costal cartilage this is very intense; as the stethoscope is carried towards the apex, the sound is found to become less loud, and probably at the apex itself it ceases to be audible. On auscultating in the reverse direction the bruit is loudly heard in the direction of the aorta and the carotid arteries. In some cases it may be heard at the back, but then only about the level of the spines of the scapulæ.

Flint considers that an aortic systolic murmur is loudest at the second right intercostal space close to the sternum. Hayden considers that its maximum is at midsternum; exceptionally it is audible at the back in the left interscapular space. Guttmann says that it is essential "in investigating any case of aortic disease, that the *whole* of the sternum should be carefully auscultated, as the murmur presents its greatest intensity sometimes at one spot, sometimes at another, on the surface of the bone."—"Physical Diagnosis," Sydenham Society's Translation, p. 291.

In case of a murmur having these characters, if you have eliminated the probability of an anæmic causation,

you will have no difficulty in diagnosing obstruction (stenosis) of the aortic outlet due to a diseased condition of the valves. The concurrent signs will be hypertrophy, without dilatation, of the left ventricle, with small hard arterial pulse. The pathological changes which give rise to this form of murmur consist in thickening, rigidity, and fusion of the segments of the semilunar valves of the aorta, so that the orifice of exit of the blood from the left ventricle is narrowed. This may occur to such extent that the orifice will barely admit a probe. Sometimes the valves are hardened from atheroma and calcareous deposit, sometimes fringed and obstructed by fibrinous vegetations or warty excrescences, sometimes roughened by ulceration.

It is important, even from a diagnostic point of view, to attempt to define under what conditions these various causes of obstruction of the aortic orifice arise. A significant and somewhat strange argument meets us at the very commencement of the investigation. Taking the positive evidence of post-mortem examinations, lesions affecting the aortic valves alone are of very common occurrence. Dr. King Chambers, in analysing 367 cases in which valvular lesions of the heart were discovered at the autopsies, found that the aortic valves were solely affected in 107, the mitral only in ninety-six. The general experience of observers has been that the frequency of disease in the mitral valve alone, and at the aortic orifice alone respectively, is about equal, but that the proneness of the mitral is slightly the greater. Compare this observation with clinical experience; I think you will find all physicians concur in saying that mitral lesions *per se* are diagnosed far more frequently than aortic. Of the hundred cases of heart disease which I mentioned

in an early part of these Lectures, as observed by myself, only fifteen were declared by the physical signs to be solely due to morbid changes at the aortic orifice, whilst no less than fifty-eight were mitral. It seems fair to conclude from these facts that aortic lesions are often present during life, but are only discovered after death. The regurgitant lesions which we shall soon consider, are much more baneful in their *obvious* effects than the obstructive lesions of the aortic valves. It is very probable, therefore, that obstructive lesions are more frequently present than detected. Positive experience confirms this view in many cases, especially of sudden and alarming symptoms in old people with cerebral or visceral disease; patients who are sometimes brought moribund to the hospital are found to present obstruction of the aortic outlet. Now as to the forms of disease which produce aortic obstruction. Of twenty-four cases observed by myself in which the physical signs indicated aortic obstruction, ten were obviously rheumatic, nearly all having suffered well-defined rheumatic fever. Flint records that of thirty cases of aortic lesions, rheumatism had occurred in sixteen. We shall see, however, that the aortic valves are less prone to be attacked by rheumatic endocarditis than the mitral. Rheumatic endocarditis attacks first the mitral valve, then extends upwards involving the endocardium lining the ventricle, till it includes the aortic valves in the morbid change. In the majority of cases in which we hear an aortic obstructive murmur without evidence of impairment of the mitral valve, endocarditis has not been limited to the aortic valves, but has begun in the mitral, though its effects have not been sufficient to destroy the integrity of the latter.

Besides the alteration of the valves from rheumatic endocarditis, there is another cause which is very common in inducing obstruction—atheroma. In this the valves are often incrusted with calcareous deposit. This form of alteration is exclusively met with after middle age, and is by far the most common cause of aortic obstruction in patients past the prime of life.

The most common cause of mechanical impediment at the aortic orifice is not primary disease of the heart or of its valves, but of the *aorta* adjacent to the valves. The latter become implicated by extension of the disease. The morbid process (*aortitis; endarteritis*) commences by swellings, localized in patches, in the internal coat of the vessel, with infiltration of the layer next to the endothelium by cellular elements; subsequently such infiltration extends outwards to the muscular coat. In the later stages degeneration occurs, so that the elements become yellowish and softened from fatty change, or undergo calcareous transformation, giving rise to hard stony plates in the wall of the vessel. This stage of degeneration, fatty or calcareous, is the condition known as atheroma. Occurring in the neighbourhood of the aortic semilunar valves, these are involved in the thickening; so they may become of fibro-cartilaginous consistence, or hard and bony from calcareous transformation. The conditions leading up to such morbid change in the arteries are probably (*a*) alcoholism. Dr. Hayden believes that amongst the humble classes alcoholism is the chief cause of the diseases of the aortic valves—that is, it induces the lesion of the aorta, which involves the valves; (*b*) gout or the lithic acid diathesis. This, according to the same authority, is the most frequent disposing cause amongst the rich. The acute and sub-acute forms of rheumatism do not predispose to the affection, whilst the gouty forms, such as chronic rheumatic arthritis, are distinctly associated with the arterial disease; (*c*) syphilis. I have had abundant evidence, to my mind conclusive, that endarteritis and aortitis can be the direct result of the action of the syphilitic poison; (*d*) muscular strain. This has been strongly insisted upon by Dr. Clifford Allbutt, who has pointed out the proclivity to aortic disease of persons whose avocations subject them to severe or sustained

effort, such as soldiers, strikers, lifters, &c. I confess I am not convinced that mere muscular effort can induce the disease in a *previously healthy* aorta. In the cases I have observed, when the affection existed in the classes mentioned by Dr. Allbutt, there has been a strong suspicion that a concurring cause might have been alcohol or syphilis, or both combined. At all events, effort is a proximate cause, and with it may be classed constriction of the chest by tight clothing, &c.; or a condition of persistently high tension in the general arterial system. This is most typically expressed in chronic renal disease, where there are tight arteries and hypertrophy of the left ventricle. I entirely agree with Dr. F. A. Mahomed, who has called attention to the fact that this tendency to high arterial tension may be manifested in the early ages of life, and when there is no renal implication whatever. It is, therefore, of the highest practical importance to recognize it, to caution the subjects of it against any possible overstrain, and so to ward off the actual disease of the arterial coats, to which it tends. In these cases the pulse may be recognized as hard and full by the finger, and there may be signs of some hypertrophy of the left ventricle, with heightened tension in the aorta, as shown by accentuation of the aortic second sound; but there is no evidence comparable in value with that obtained by the sphygmograph (see Part II.) If at any age a condition of high tension in the arteries is demonstrated, the danger of disease in the arterial coats should be recognized.

For the differential diagnosis of the rheumatic and the atheromatous forms of disease occasioning obstruction at the aortic outlet, the evidence afforded by the sphygmograph is of great value. The tension in the atheromatous form is much greater than that which usually obtains in the rheumatic; in the former is found the typical trace of aortic obstruction. See typical tracing of aortic obstruction in Part II.

By both these forms of disease (rheumatic endocarditis and atheroma) the narrowing of the aortic orifice may be extreme, and yet the signs of subjective and objective may not be pronounced. In two cases, one mentioned by Stokes and another occurring in

America, the orifice left in the fused and hardened valves was so small as only to admit a small probe, and yet disease of the heart was not suspected till the occurrence of acute disease of the lungs which proved fatal. If the signs in such extreme obstructions are obscure, they are still more so in the form of endocarditis characterized only by vegetations upon the valves. According to French observers, these lesions have nothing to do with rheumatism, but are associated with various maladies (Lancereaux, "Anatomie Pathologique," p. 220). It is this form of endocarditis which is especially associated with chorea in young subjects, and with the accidents of cerebral embolism in the old. There is yet much obscurity as to its pathogenesis; it is characterized by hyperplasia of the superficial layer of the endocardium, giving rise to little excrescences forming groups upon the ventricular surface of the valves, sometimes fringing their free borders. These vegetations are often attached to the surface of the valve by slender pedicles easily detached; their size is frequently augmented by the attachment of fibrine derived from the blood current. It differs in histological characters from the rheumatic form of endocarditis which affects the deeper fibrous structures of the valves and involves their whole substance. It seems very probable that the French observers are correct in differentiating this form pathogenetically. Lancereaux says that it may occur under various obscure conditions, but especially in alcoholism, the puerperal state, and, perhaps in intermittent fever. Whether isolable or not, we must admit that this form of endocarditis may accompany other forms—in fact that, though it may probably arise *per se* in certain as yet untraced conditions, it

frequently occurs upon any valve which has undergone morbid change from other causes.

Another form of disease affecting the aortic valves, which, though rare, must be borne in mind as affecting diagnosis, is that in which the valves are *ulcerated*. Under certain circumstances of depressed vitality, the valve already diseased tends to ulcerate; the patient is seized with rigors and symptoms of septic poisoning. The débris of the ulcerated portions of the valves form plugs which, being carried by the current of blood, cause embolism of many arteries throughout the system as well as general blood-poisoning. In eleven cases recorded by M Lancereaux, the aortic valves alone were affected by the ulceration in six. The disease may arise in various adynamic conditions; several cases have been recorded as occurring in the puerperal state. You may conclude that when in a case of aortic obstructive disease (mitral conditions we shall consider hereafter) your patient is seized with severe rigors, with pyæmic symptoms, with vomiting and diarrhœa, with alternations of very high with low temperatures, and especially with signs of cerebral embolism, there is present an ulcerative endocarditis. The disease is uniformly fatal.

To resume, concerning aortic obstructions in general, especially as regards prognosis. When you have a loud obstructive murmur with a history of rheumatism or of senile change, with some cardiac hypertrophy, the lesion, compensated as it is by increased force of the heart, need not give occasion to a grave prognosis. The patient may with considerable probability live long in spite of the obstruction. In the case of the soft murmur you should be guarded in your prognosis. Examine your patient with great

care, for these cases are often overlooked. Remember in children the proclivity to chorea; in advanced age, in puerperal conditions, in alcoholism, and in Bright's disease, the danger of embolism. When you are called to a case of so-called apoplexy where the symptoms occur *very suddenly*, always carefully auscultate the heart, remembering the great probability that there has been cerebral embolism by detachment of a vegetation from a diseased valve.

We return now to the aortic cartilage. Suppose that we find the first sound to be unaccompanied by murmur but not so the second or diastolic sound. The latter, which should consist only of the click occasioned by the closure of the semilunar valves, is complicated by a murmur. Like the systolic, which we have just considered, this may be soft and short, or loud and pronounced. If soft, you will notice first, its point of greatest intensity. You will probably hear it at the aortic cartilage, but on carrying your stethoscope to the left side of the sternum and auscultating over the cartilage of the third or fourth rib, it will be yet louder. The reason of this we shall presently see. Notice in the next place whether some of the clicking sound of the closure of the aortic semilunar valves is heard with it—*i.e.*, whether it *accompanies and not replaces* the aortic second sound; if this occurs, it is evidence that some of the segments of the valve are capable of performing their functions—that the lesion does not involve them all.

This second sound murmur, instead of being soft and local, may be loud and prolonged; in quality it may be rough, or musical, and it may be heard over a wide area. You may not hear it over the right or left base, or up the great vessels, but by carrying

your stethoscope downwards along the centre-line of the sternum you arrive at a spot where the murmur with the second sound is distinctly heard. Exceptionally it is heard only as you get near the apex of the heart. You have many collateral signs to guide you in the diagnosis: the visible throbbing of the arteries, the heaving of the præcordial region, the signs of hypertrophy and dilatation of the left ventricle. In no condition is the probability greater of enlargement of the heart; in some cases there is enormous increase of mass and weight, the so-called "cor bovinum."

There is another objective sign which I take to be of great value. On making an *ophthalmoscopic examination* of the *retinal vessels* by means of the erect image, you observe *pulsation of the veins or arteries, or of both.* Dr. Stephen Mackenzie has done great service in drawing attention to this sign. I have made ophthalmoscopic examinations in a large number of cases in which an aortic second sound murmur existed, and I I have rarely failed to find visible pulsation of the retinal vessels. Such pulsation is found most commonly in the veins, but both arteries and veins pulsate in many cases. This sign may have great value, especially when other signs are masked—for example, when the lungs are affected, and the loud rhonchi and râles of bronchitis render it difficult to hear the sounds of the heart.

Suppose that you have heard a second sound murmur, and that by its position and by the existence of collateral signs you have located its production in the aortic outlet, what is its pathological significance? It means that there is regurgitation of the blood-stream into the left ventricle when the heart is in diastole, on account of the imperfect closure of the

valves which guard the aortic outlet. In the case of the systolic murmur, we had *obstruction* afforded to the onward stream of blood through the aortic orifice; in the case of this diastolic murmur we have *insufficiency* of the valves to close the orifice after the systolic gush is over.

Imperfection of the valves, permitting regurgitation into the left ventricle, is brought about by pathological processes similar to those which induce obstruction. In the rheumatic form of endocarditis the valves, after having become thickened by hypertrophy of their connective tissue, undergo a slow and gradual process of contraction, so that the free edges of the valves are retracted from the centre, and a gap of necessity results. Or, in the villous form of endocarditis, a bunch of vegetations may depend from the segments of the valve on their ventricular aspect, and so weigh down such segments as to prevent their apposition in diastole. These masses of vegetations may vary in size from a pin's head to a walnut. Again, such imperfection of the valves as may induce regurgitation, may be caused by the rigidity of segments which have undergone atheromatous change. In rare cases the valves may become ruptured through violence or perforated by ulceration.

In all these pathological conditions, the mechanical effect is reflux of blood, in diastole, through the abnormal gap in the valves. The secondary effects are, first, that there is a deficiency of supply of blood to the general arterial system; secondly, that the left ventricle contains always too much blood. The further consequences are, that the ventricle undergoes compensatory hypertrophy, thus making up for

the loss to general arterial system induced by the reflux, and that, from its over-repletion (for you will understand that it contains blood early in diastole when it ought to be empty, and the normal amount of aërated blood is superadded to the amount which has regurgitated through the imperfectly-closed aortic orifice) the ventricle becomes dilated. You will thus comprehend why, in the case of aortic obstruction, we have hypertrophy only of the left ventricle, whilst in aortic regurgitation we have hypertrophy and dilatation. In obstruction there is enhanced power of the ventricle to overcome the difficulty, but no distension of the ventricle by an abnormal content of blood.

You are now in a position to understand the mechanism of the murmur of aortic regurgitation—it is the murmur caused by the backward rush of blood through the partially-closed and imperfect aortic orifice. Remember that this is not a merely passive reflux—it is not simply by the weight of the column of blood that the murmur is occasioned. The ventricular systole drives the blood into arterial channels which are both elastic and muscular. There is a recoil, therefore, of all the arteries which have been distended by the ventricular systole, and the blood is forcibly urged backwards into the ventricle. Thus you may understand how prolonged and loud the murmur is in some cases.

Aortic regurgitation is most commonly met with at or after the prime of life (of fifty cases noted by Von Bamberger, only fifteen occurred before the age of thirty), and in the male sex, the proportion in recorded cases being three males to one female. The prognosis is generally bad; in the rare cases of youthful patients, compensation by hypertrophy of the ventricle

may be such that but little trouble is experienced,* but when adult life is attained the lesion is one of the gravest that can affect the heart. This is one of the conditions in which *sudden* death may occur, and the subjects should be cautioned against excitement and over-exertion.

Suppose, now, that you hear two sounds at the aortic cartilage, the one with the systole and the other in the diastole. There is a *double murmur*. Or over certain spots whereto, as I have described, an obstructive "bruit" may be carried by conduction or convection, you may hear a first sound murmur, and over other spots in the direction of the retrograde current you may hear a second sound murmur. You may conclude in such cases that there is a combination of the two lesions which we have just considered—that there is aortic obstruction as well as aortic regurgitation. This is not uncommon. The semilunar valves, roughened on their ventricular aspect, or rigid and offering an obstructed orifice of exit for the blood, are also imperfectly opposed in diastole.

We have now considered three forms of heart-murmur—the aortic direct, or obstructive; the hæmic or anæmic, and the aortic regurgitant or murmur of aortic insufficiency. It will be well to make a short digression and endeavour to understand the physical cause of these abnormal sounds.

There are few subjects which have been more voluminously debated than the *physical causes of cardiac murmurs*, and few concerning which there is less accord. It would appear that each observer has taken infinite pains to elaborately convey his impres-

* I have met with several instances of the affection in children, who nevertheless evinced little or no cardiac distress.

sions, with the result of convincing no one but himself. No two observers seem to be in complete agreement. It will serve, I think, no good end to make a critical examination of the various theories which have been propounded; I purpose, therefore, to express my own views on the subject in a way which I conceive to be the most simple, the most practically useful, and the most in accord with the present teachings of science.

1. Sound is the effect of vibrations transmitted to the organ of hearing. The sounds of the heart, normal and morbid, are due to vibrations conveyed from the surface of the thorax to the ear. If the ear be applied closely to the chest (immediate auscultation), the vibrations of the thoracic wall are communicated to the intervening layer of air, and then to the external ear. If the stethoscope be used (intermediate auscultation), the vibrations are transmitted to its walls, and by these to the air intervening between the ear-piece and the tympanum. The tube of the stethoscope does not perceptibly weaken the sound. "The law that the intensity of sound increases in inverse proportion to the square of the distance, does not apply to the case of tubes, especially if they are straight and cylindrical."* Many vibrations are communicated to the chest-wall, from the movements of the heart, and the fluids and structures in relation with it, which are not capable of giving rise to impressions of sound. Such are impulse and thrill. To be perceived by the auditory centre, vibrations must have a certain rapidity and a certain amplitude or intensity. The tactile faculty

* Ganot's "Elementary Physics," translated by Atkinson. Eighth edition, p. 177. London: Longmans.

can appreciate vibrations which are too slow for the auditory, and the auditory those which are too rapid for the tactile; but the areas of perceptibility by the two faculties overlap, so that vibrations may in some cases be detected by both and in others tactile impressions may be supplementary in duration to auditory or *vice versâ*. Thrill may precede, accompany, or succeed murmur. It is to the thoracic surface, then, whereto vibrations are conducted, some of which are sonorous, or capable of exciting auditory perception. It is at the thoracic surface alone that the conditions obtain for the production of sound, for it is here only that the vibrations are given to the air.

2. The sounds heard are produced by vibrations communicated to the air by the thoracic wall, irrespectively of the mechanical means by which such vibrations are produced. This proposition is proved by the telephone. By it sounds are not transmitted, but vibrations similar to those of the sounds are produced, and the effect on the organ of hearing is the same in the one case as in the other. It is further demonstrated by the phonograph, by which sonorous vibrations are made to record themselves as motion-vibrations upon the surface of a soft metal; these being by mechanical means recommunicated to the air, the original effect on the organ of hearing is reproduced. There is synthesis of sound from the mechanical vibrations. It appears to me, therefore, that the *quæstio vexata* of the site and mode of production of *sounds* in the heart, the vessels, or the fluids, is a barren controversy. Sounds are not produced, save at the thoracic wall, where the air receives its undulations. In the heart and its surroundings are produced *vibrations*, which at the sur-

face may, or may not, give rise to impressions of sound.

3. The normal sounds of the heart partake rather of the character of *noise* than of music; they are either sudden and abrupt, or dull and muffled. The second sound is the uncomplicated sound of valve-tension, such as one imitates by suddenly stretching a previously lax piece of membrane. The sound is of too short a duration to be capable of musical determination: the auditory faculty receives it as a shock. The first sound is for the most part similar to the second—that is, it is chiefly due to valve-tension occurring over a larger area (in proportion, as the mitral and tricuspid valves combined exceed in extent the aortic and pulmonary), but modified first, because the intervening walls of the ventricles subdue the sound in proportion to their thickness; secondly, because the (muscular) sound of the contracting ventricles supplements and prolongs the valve-sound. The muscular sound is dull, because the fibres mutually interfere to arrest their own vibrations.

4. The morbid sounds which are heard in relation with the heart and vessels, partake, on the other hand, rather of the character of *music* than of noise. They are not abrupt, but, varying in intensity and pitch, commence at one end of the scale as a scarcely perceptible blowing (aspirate), and become rough and grating, or else occur in many varieties of pitch up to the production of a distinct musical note.

5. The organic conditions giving rise to such murmurs as have their origin in disease or abnormality of the valves or orifices of the heart may be for the most part referred to two types: first, in which the orifice which originates the vibrations occasioning the

murmur is converted into a *ring;* secondly, in which across the orifice and in the blood-stream there is a vibrating *tongue*. In these conditions a strict analogy obtains with musical instruments. In one case the fluid which is the intermediate agent of vibration is the blood, in the other it is air. The first type is exemplified by the flute, the cornet, or other usual form of wind instrument; in these, vibrations are produced by the forcing of air, under compression, against the wooden or metallic walls of the instrument. More familiarly still, it is illustrated by whistling produced by the mouth with the lips pursed up so as to make a circular aperture, the pitch of the note produced being heightened in proportion to the decrease in size of the aperture, and the intensity or loudness varying in proportion to the force of expiration. It seems to me a popular error to ascribe too much of the sound-producing agency to currents of air forced through the aperture. The air is rather the intermediate than the immediate agent—that is, the force of expiration sets the solid boundaries of the orifice vibrating, these vibrating solids communicate their vibrations to the air, and the stream which issues from the orifice has little to do with the production of sound-vibrations. This can be easily proved. Produce with the mouth an audible "whistle," then apply the fingers to the pursed-up lips so as to stop their vibrations, but in such manner that the exit of air from the orifice is unimpeded. You will find that though the air issues just as before, the "whistling" sound is arrested by the arrest of vibration of the walls of the orifice of exit. A similar experiment with a metallic whistle has the same result. So I think we may infer that the vibrations which give rise to sound in the

case of the intra-cardiac murmur start from the solid structures rather than from the fluid blood, though this latter is an agent for the communication of vibrations *to* the solid structures. Savart's fluid veins are agents for the production and communication of vibrations, which imparted to solids give rise ultimately to sonorous vibrations. The second type of murmur is illustrated by the Jew's harp or accordion, in which a tongue of metal is set in vibration. At the aortic orifice it is exemplified when a shred of fibrin, a prolongation of fibro-cartilaginous material or the débris of a ruptured valve stretches across and vibrates in the blood-stream. When so originating, the murmur often has a markedly musical quality; it usually is diastolic, but may be systolic, and is not unfrequently double.

5. We have already seen (p. 96) that the sounds heard over the heart-region are not immediately superficial to the positions whence they originate—that is, the vibrations which give rise to sonorous impressions are conveyed from their sources to certain areas of the thoracic surface. Such transmission occurs in two modes (1) by *conduction*, so termed when the vibrations are conveyed by *still* media or solid structures. The normal lung, filled as it is with air, is a very bad conductor of vibrations; when, however, there are condensations of the pulmonary tissue, exudations or solid growths, these may act the part of conductors, and the heart sounds, normal or abnormal, may be heard in unusual situations. In the case of the diastolic aortic murmur, the sternum, which is comparatively a good conductor, causes the sound to be heard in a direction down its central-line or along its left border. (2) There is, however,

another way by which vibrations are transmitted, that is, by media in *motion*. A murmur is best heard in the direction of the current of blood. This was called by the late Dr. Hyde Salter the law of *convection of murmurs*, and explains away many difficulties. This also may be illustrated by analogy with the air. On a still day everything may be silent, but let a breeze spring up, and the sounds —of distant bells, for instance—are wafted to the ear. The sound is heard only in the direction of the current of air ; if the wind change it will be no longer audible. In like manner, a cardiac murmur is heard in the direction of the current of blood. In the murmur of aortic obstruction, the vibrations generated at the time of the systole are carried by the blood-stream into the aorta and up the great vessels. In the murmur of aortic regurgitation, the direction of the current of blood at the time of occurrence of the sound is exactly the reverse of the former ; the stream is gushing back into the left ventricle, and the line of convection is downwards from the aortic cartilage to the apex of the heart. In some exceptional cases the regurgitant murmur is only heard at or near the apex.

The late Dr. Hyde Salter called attention to the fact that a diastolic murmur generated at the aortic outlet was in exceptional cases *audible only at the apex of the heart*. Two illustrative cases were quoted (*Lancet*, August 14, 1869, p. 225). In one of these there had been, no doubt, rheumatic endocarditis :—"It was quite certain that the murmur so plain at the apex and inaudible at the base, was due to aortic regurgitation—that is, that the stream of blood flowing down towards the apex carried thither the sonorous vibrations of which conduction failed to give any evidence in the immediate neighbourhood of their production." In the second case there was "a very distinct diastolic mur-

mur" heard at the left apex in the case of a female in whom it was extremely probable that there had been *rupture of the aortic valves.* These observations have been confirmed, amplified, and rendered of great practical importance by Dr. Balthazar Foster (see "Clinical Medicine," p. 112 *et seq.* London, Churchill, 1874). Dr. Foster's observations were also in cases of rupture of the aortic valves, and the experience of one case dictated a precise diagnosis in another. When a diastolic murmur of aortic origin is conducted to the left apex, "it depends on the regurgitation taking place through incompetency of the *posterior aortic segment*, either at its right angle or through perforation of its curtain." In such pathological conditions the regurgitant blood-column falls upon the upper segment of the mitral valve; this was proved, not only by experiment after death, but by inference from the thickening of the segment which had probably taken place on account of the stream which had been projected against it during life. The vibration produced, notwithstanding that the lesion is aortic, being of the mitral valve, is heard in the mitral area—that is, at the apex. Dr. B. Foster adds:—"I believe we may also say that a similar murmur propagated towards the ensiform cartilage indicates incompetency of either the left or the right coronary segment, by which the regurgitant current is thrown more upon the septum of the ventricles."

The diagnosis of rupture which may occur in the case of previously healthy aortic valves is to be made from the history of the onset of symptoms:—sudden pain at the præcordium, palpitation and dyspnœa arising in direct relation with violent strain, effort, or after a blow upon the sternum, in the absence of a history of rheumatism, but attended with the physical signs of aortic regurgitation, or of aortic obstruction and regurgitation combined. Dr. Hayden adduces also the existence of *systolic arterial thrill* as an important sign for the physical diagnosis of such cases.

Supposing Dr. Foster's experience to be confirmed, it becomes possible to determine in cases of aortic regurgitation which segment is affected, and this may be of high importance as regards prognosis. "Two of the segments have above them each a coronary artery which is filled by the blood column as it rebounds from their curtains. When these segments are torn down and retroverted, the regurgitant blood-column running

past the mouths of the coronary vessels must to some extent diminish the amount of diastolic blood-wave which they receive, and consequently impair the heart-nutrition. . . . That segment of the aortic valves by whose incompetency, we believe, a murmur is specially carried to the left apex, has no coronary artery above it, and therefore when it is affected, we should expect the coronary circulation to suffer less than when either of the other segments is imperfect." Death usually follows rupture of the aortic valves within three months, the maximum duration of life has been four years and a half. In the case of injury to the non-coronary segment recorded by Dr. Foster, life was prolonged to nearly three months; whilst the duration was eighteen months, and three months in the other cases. It would appear, therefore, that when in a case of aortic regurgitation a diastolic murmur is localized at the apex of the heart, it is probable that the posterior segment of the aortic valve is affected, and that the chances of duration of life are greater than when the murmur is heard at the usual situation over the sternum.

6. Vascular murmurs—the so-called hæmic murmurs—are due to vibrations produced in the walls of vessels. The murmur heard over the site of the aortic valves is the effect of vibration of the initial portion of the aorta; sounds of similar character heard over the carotid and subclavian arteries are due to vibrations of the arterial walls occurring in certain areas. It is characteristic of these murmurs that they are not transmitted along the vessel in unbroken continuity, but are heard over certain portions of it; they are much intensified by a little pressure with the stethoscope. Murmurs closely resembling these may be produced by such pressure even in conditions of health. Vascular murmurs may be heard in acute febrile diseases, but they are especially associated with anæmia and chlorosis. It has been proved, both by Marshall Hall and by Hope, that such murmurs can be induced in animals by copious bleed-

ings, and we are familiar with the fact that they appear in the human subject after sudden and copious hæmorrhage. That they are not due to the mere abstraction of *quantity* of blood, however, is proved by the experiments of Richardson, who found that the "bruits" were produced in dogs after injection of large quantities of water into the veins. Moreover, in common with other observers, I have noted them in cases where there was good volume of blood in the arteries. There can be little doubt that a watery condition of the blood aids to the production of such murmurs; and this is only probable, for a mobile fluid can oscillate much more readily than a viscid one. Neither decrease in quantity nor deterioration of quality in the blood can, however, be the only and absolute cause of the murmur, for the latter is not constant in conditions of anæmia, nor is it heard throughout the great arterial vessels. The localization of the murmur points to a local cause, and this cause I take to be a modification of tension in the arteries in certain areas, whereby the walls of the artery in these portions are rendered prone to vibrate. These murmurs are particularly marked when there is any cause for the abnormal conduction of vibrations to the surface of the chest. This is especially the case in subclavian murmur, which is thus of diagnostic importance in the early stages of disease in the apices of the lungs. The venous hum—*bruit de diable*—has its origin in the internal jugular vein. It is due to the vibration of the coats and valves of the vein, just as the arterial murmur is due to the vibration of the walls of the artery, such vibration being communicated by the whirling movement of the blood. "The blood flows from the relatively narrow

jugular vein into the relatively wide bulb (the part at which the vessel debouches into the innominate vein), and is thus caused to sweep in a somewhat spiral course round the walls of the chamber, so that the mode of origination of the venous hum may be regarded as strictly analogous to that of murmurs in the arteries. This dilatation at the end of the vessel remains permanently wider than the upper part of the vein, as its sides are held apart by the tense cervical fascia."* The venous hum is intensified when the patient turns the head *from* the side which is auscultated; thus, the superficial structures of the neck being put upon the stretch, the jugular vein is compressed and narrowed. It is obscured or extinguished when the patient resumes the recumbent position, the aid to the force of the current afforded by gravity being thus removed. The hum is generally louder on the right side of the neck.

Chauveau and Corrigan have both demonstrated experimentally that "when a liquid current is hurried past a constriction in its passage into a wider space beyond, a ripple or perturbation in the current is created, giving rise to vibration and murmur."† This I consider to be the key to the explanation of vascular murmurs, both venous and arterial, only I do not attribute the sound-producing vibrations to the liquids, but to the solids, to which the vortiginous movements of the fluids impart motion. In the case of the basic aortic murmur, there is a condition of impaired tension at the origin of the vessel, so that its walls vibrate. In the case of arterial

* Guttmann's "Handbook of Physical Diagnosis," p. 308.
† Hayden's "Diseases of Heart and Aorta," p. 250.

murmurs there is non-equable tension of the vascular wall, and at the portions where tension is impaired, eddies are produced which set these portions vibrating. The venous hum is the effect of deficient impletion or defective muscular tone in the jugular, whose walls vibrate owing to the eddies developed in its fluid contents.

LECTURE VII.

AUSCULTATION.

PART III.

Hæmic pulmonary murmur—Pulmonary stenosis—Relation to cyanosis—Tricuspid regurgitation—Ventricular murmurs—Murmurs in chorea—Myocarditis—Arguments concerning cause of non-valvular murmurs—Cardiac phenomena of typhoid fever—Organic mitral murmurs—Causes of mitral lesions—Varieties of lesion—Mitral insufficiency—Mitral stenosis.

We now leave altogether the region of the aortic valves and cross the sternum to the second left interspace and the sternal end of the third cartilage—the area of the valves of the pulmonary artery.

Suppose that you hear a soft murmur with the first sound localized in the pulmonic area. The chances are enormously in favour of this being inorganic—vascular. Observe all the rules as regards differential diagnosis which I have given you, for the discrimination of anæmic murmurs when heard at the aortic base. Especially notice whether the sound is carried along the left subclavian. If it be strictly localized, the hæmic murmur may be generated in the pulmonary artery. Dr. Flint considers that an inorganic murmur emanates as frequently from the pulmonic orifice as from the aortic. I must say that I have not observed this in my own experience. I have found the soft murmur heard at the left base in the majority of cases, to be distinctly traceable to the left

subclavian artery. I would say of a soft systolic murmur localized in the pulmonary area, always assume it to be inorganic or independent of structural change, unless there is strong collateral evidence in favour of its being organic.

Assuming that you have eliminated anæmia as a possible explanation of the murmur, you have yet to consider whether the sound may be produced by the pressure of a tumour upon the trunk of the pulmonary artery. It has been said that tuberculous lung can give rise to such pressure. I should explain the murmur heard in these cases as due to the conduction by the tubercular consolidations of a hæmic murmur generated in the pulmonary artery. Tumours in the mediastinum, enlarged bronchial glands, cancerous and other growths in the lung may undoubtedly cause pressure against the trunk of the pulmonary artery, and give rise to a systolic murmur. All such probabilities of extra-cardial causation you must eliminate by careful examination.

We will imagine that you have eliminated anæmia and pressure on the pulmonary artery as probable causes, and yet you hear a localized murmur over the situation of the pulmonary semilunar valves. This murmur with the first sound may be soft, and heard only at the spot indicated, or it may be loud, and heard over a wide area, but distinctly most intense about the third left cartilage. You may be sensible that it is very superficial—generated very close to the ear. A murmur with these characteristics will, with much probability, be due to *obstruction of the pulmonary artery*, but before completing the diagnosis we will briefly consider the pathological causation and clinical concomitants of such a condition.

In considering aortic disease, I made no mention of congenital defects, because these so rarely affect the aortic orifice that they do not practically influence diagnosis. Aortic defects are not congenital, but acquired. It is quite otherwise with pulmonary lesions. Intra-uterine malformations are far more likely to involve the pulmonary artery; moreover, when endocarditis attacks the "fœtus in utero" it is the *right chambers* of the heart that are much more frequently affected. The rule in after-life is exactly reversed.

The pathological causes which give rise to obstruction of the pulmonary orifice operate chiefly in fœtal life. Through faulty development, the pulmonary artery itself may be contracted in varying degree, even to complete obliteration, a blind extremity or rudimentary cord only remaining. Or the semilunar valves may be fused together, and may form a membranous, cartilaginiform or cretaceous septum with a circular or slit-like opening. Or the contraction may be below the valves at the apex of the right ventricle —the conus arteriosus dexter—the cause being the shrinking of the muscular tissue, subsequent to inflammation (myocarditis). In one or two cases vegetations like those described in case of aortic disease have been found about the valves. In the rare cases met with as originating at or after adult life atheroma may be a cause, or an inflammation due to direct violence, to which the right ventricle and pulmonary artery, by their superficial position, would seem to be more liable.*

For convenience in clinical diagnosis, I think it best to group the cases according to age.

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* Von Dusch, "Lehrbuch der Herzkrankheiten." Leipzig, 1868, p. 248.

In infancy the patient will probably be the subject of cyanosis, presenting the characteristics which we have already considered. You will hear the murmur loudly at the base, though in the small area of chest presented in infancy you may not be able precisely to locate it in the pulmonary region. The blueness of surface, however, the venous distension, and the extreme improbability of aortic disease at this period of life, will leave little doubt as to the diagnosis. You may safely infer that a patent foramen ovale, or an aperture in the interventricular septum, co-exists with the pulmonary obstruction, for such is the case almost invariably.

In later childhood you will have more chance of localizing the murmur, unless it be exceptionally loud. You must bear in mind, however, that aortic disease may possibly have been developed by rheumatic endocarditis subsequently to birth; therefore the differential diagnosis between aortic and pulmonary obstruction is now necessary. If the bruit be pulmonary, you will probably observe a condition of cyanosis, at least intermittingly, and venous turgescence increased by exertion and by coughing. Moreover, you may be able to ascertain that the right side of the heart is hypertrophied and dilated. There may be venous pulsation. The following case, which occurred under my own observation, affords many points of interest:—

A little girl (L. S.), aged eight and a half, was admitted under my care at the North-Eastern Hospital for children, on January 12, 1873; she seemed weak, and was *excessively pale*, but presented no blueness nor obvious dyspnœa. On auscultating the heart-region a very loud rough first sound murmur was heard at

the base, quite as intense at the aortic as at the pulmonary point. The child had been ailing occasionally ever since birth, but there was no obvious symptom except weakness. She was one of seven children, of those, four were living, one had been stillborn, and one died during dentition. The mother was healthy, and could give no account of "maternal impression." I could not help giving a very doubtful diagnosis—it appeared to me that it was quite as probable, from the physical and general signs, that the obstruction was seated at the aortic orifice as at the pulmonary. As the case progressed, the next observed phenomenon was *diarrhœa*, which began on January 13, and became very persistent and uncontrollable. On February 1, hæmorrhoids were noticed. Progressive enfeeblement occurred, the radial pulse became scarcely perceptible, the hands very cold, while the feet were fairly warm. A week afterwards delirium was manifested; the diarrhœa persisted, and there was much abdominal pain; emaciation continued, the pallor increased, but throughout there was no cyanosis. The child died on March 10. At the autopsy we found the lowest lobe of the right lung thickly studded with masses of soft *tubercle*, varying in size from a small pea to a large bean. The left lung also was tuberculous at the apex. The large intestine was ulcerated throughout its whole length, and the mesenteric glands were a complete mass of hard tubercle. We found about an ounce of pale yellow serous fluid in the cavity of the pericardium, and the heart itself very small, pale, and contracted. The aorta and its valves were quite normal, but the pulmonary artery was very small in calibre (diameter four-tenths of an inch), its walls firm and inflexible, so that it resembled

the aorta or a systemic artery. It was a pulmonary artery in miniature, with valves minute but perfect. The area of a section across the aorta compared with that of a section across the pulmonary artery was in the proportion of three to one. The wall of the right ventricle was greatly hypertrophied, so that it was thicker at its thickest part than any portion of the left ventricle. The foramen ovale was patent, the aperture being circular, with rounded edges, the communication between the auricles quite unimpeded. There was no imperfection of the septum between the ventricles: the valves were all healthy.

This case shows that we may have a loud murmur of obstruction at the pulmonary orifice without cyanosis. The signs strongly suggested aortic constriction, but there was one chain of circumstances that led up to the diagnosis of pulmonary lesion—the occurrence of general tuberculosis. The persistent uncontrolled diarrhœa and wasting seemed to indicate tubercular ulceration; the post-mortem examination showed tubercle abundantly scattered throughout the body. Now tubercular changes in valvular diseases of the heart, are, for a reason which I will not stay to discuss for it is yet very obscure, very rare. The great exception to this immunity is in pulmonary obstructive disease. That congenital pulmonary construction predisposes to tubercle has been noticed, especially by Lebert and Peacock; the observation is valuable as an element of diagnosis.*

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* I have discussed the subject of Pulmonic Murmurs in Children more at length in "Clinical Lectures on Diseases of the Heart in Children."—Vide "*Medical Times and Gazette*, September 6, 1879, p. 255.

When adult life is attained, patients who present signs of obstruction at the pulmonary orifice rarely come under our notice. Besides the signs I have already given you, enlargement of the right chambers of the heart may now be more decided, or more readily detected; in addition, a pronounced superficial systolic *thrill* may be felt over the pulmonary area. There may be considerable difficulty in determining whether the pulmonary stenosis be congenital or acquired. Though the congenital malformation is so fatal that only about fifteen per cent. of the subjects reach the age of twenty, some cases attain to a considerable age. Instances are recorded, where the defect was undoubtedly congenital, in which the subjects lived to the age of forty (Kussmaul), fifty-seven (Peacock), and sixty-five (Stölker), respectively. In nearly all cases, however, inquiry will elicit the fact that there has been some respiratory trouble, or some tendency to lividity from infancy or early childhood. On the other hand, in acquired obstruction there may be a history of comparatively recent development of symptoms; a blow may have been received upon the præcordial region which has set up myocarditis, or in a patient past the prime of life you may observe such evidence of degeneration of the systemic arteries as would suggest the probability of atheromatous change in the pulmonary artery.

Murmur with the second sound in the pulmonary area, due to incompetency of the pulmonic valves, may be dismissed in very few words. It is very improbable that you will meet with an instance. I only know of one, recorded by Hope; in this, the pulmonary artery was dilated, and the apposition of the segments of the valve thus prevented.

Double murmur—that is, murmur both with first and second sounds—has been recorded in a few instances, but is very rare. Of course it indicates obstruction, combined with incompetence.

Having finished the exploration of the pulmonary valves, we now complete the investigation of the right side of the heart by auscultating the *tricuspid area.*

You may hear a soft blowing murmur with the first sound limited to the triangular surface occupied by the right ventricle, but most evident at the base of the *ensiform cartilage.* If you have made this observation with care, and have excluded the probabilities of extra-cardiac causation, you will conclude that the murmur is due to *regurgitation* through a defective tricuspid valve.

Remember that the conditions in regard to the production of murmur are now exactly the reverse of those which we have hitherto considered. A murmur with the first sound, the site of which is the aorta or pulmonary artery, indicates *obstruction* in one of these vessels, the systole urging the blood through the obstructed orifice. A murmur with the first sound at either of the auriculo-ventricular apertures indicates *regurgitation* through such aperture; the contraction of the ventricle whilst impelling, in the usual way, a portion of the content of blood into the vessel of exit, at the same time forces backwards, on account of the gap left by the imperfectly apposed valve, another portion into the auricle.

As regards the right side of the heart, the pathological causes which bring about this result are dilatation of the right ventricle and morbid changes in the valves themselves. Of these the more common is the

former. When from any cause there is considerable and continued venous congestion, the right ventricle becomes distended and dilated. Emphysema of the lung may dispose to this condition, but the most common cause of all is disease of the left side of the heart. Dilatation of the right ventricle follows disease of the mitral valve when there is deficient propulsive power in the left ventricle as consequence from cause. The last result of such dilatation of the right ventricle, is, that the curtains of the tricuspid valve, which under normal conditions were competent to close the auriculo-ventricular aperture during systole, are by the circumferential traction of the walls of the widened ventricle, withdrawn from the centre, so that their edges do not perfectly meet. In systole, therefore, blood regurgitates into the right auricle. The same condition may result from a contracted state of the papillary muscles to which are attached the tendinous cords of any of the curtains of the valve.

The valve may have been altered by rheumatic endocarditis, though this disease very rarely attacks the right side. It is very improbable that you will meet with a case of tricuspid murmur with a history of rheumatic origin without there being concurrent signs of disease of the mitral or aortic valves. There may, however, be a congenital affection of the tricuspid due to endocarditis in fœtal life, when disease of the left chambers is almost unknown. Rupture of the valve has also been recorded.

You must particularly note the concurrent signs of tricuspid regurgitation. These are, first, the evidences of hypertrophy and dilatation of the right side which we have already discussed—the pulsation of the right auricle may be obvious in the second right intercostal

space; secondly, turgescence of the veins of the surface and often cyanosis; thirdly, in some cases, venous pulsation evidenced in the jugulars or in the liver. The symptoms of tricuspid regurgitation are dyspnœa, dropsy, and the distresses of heart-disease in their gravest forms. For a very good account of the secondary consequences of tricuspid insufficiency, consult Dr. Milner Fothergill, on "The Heart and its Diseases."* It is very important for you to call to your aid these concurrent signs, for whilst they will assist you when you hear a murmur soft and ill-defined as it often is, they will lead you to a diagnosis when there is no murmur at all. *Tricuspid regurgitation without murmur* is far from uncommon; thus differing from mitral regurgitation, the reason being the comparative feebleness of the right ventricle.

If you have arrived at the diagnosis of tricuspid regurgitation, the prognosis is very unfavourable. There is in this condition no chance of compensating change to overcome the difficulty, such as exists in most other morbid conditions of heart. Enhanced power of the right ventricle but serves the more to engorge the venous system by the greater regurgitation; greater strength of left ventricle, if attained, would but increase by *vis à tergo* the tension in the venous system, but it is not attained because the arterial supply to the ventricle is diminished by the existing conditions.†

You may possibly hear in the tricuspid area a murmur which does *not* occur with the first sound, but immediately before it. It is a *presystolic* murmur, and indicates *obstruction* of the tricuspid orifice.

* London: Lewis, 3rd edition, p. 85.

† Cf. Von Dusch, *loc. cit.* p. 230.

This lesion, however, in the absence of disease of the other valves of the heart is almost unknown, and its consideration may be conveniently deferred until after the exploration of the mitral area.

Before proceeding to auscultate the mitral area, I shall ask you to consider certain sounds which are heard in the interval between base and apex, and even over the apex itself—sounds which are *not* due to any organic disease of the valves.

I can show you many examples in which the first sound of the heart, heard on auscultating between base and apex—that is, over the right ventricle—is *rough* in its character. The sound is not pronounced enough to be designated a murmur, but the quality of the contraction is not *pure* like that of the healthy ventricle. I have found this to be distinctly the case in some instances of dilatation of the right ventricle, such as one meets with in chronic pulmonary complaints. It occurs also in some cases of anæmia, and is, in my opinion, evidence of a weak right ventricle.

The sound may, however, be more than a mere roughness: it may be termed a soft first-sound murmur. This also may be due to anæmia, especially when the condition of corpuscle-deficiency is associated with dilated right ventricle. That anæmic murmurs are occasionally generated over the right ventricle, I have no doubt whatever. In these cases the soft murmur may be heard only as far as the base, or, and this much more commonly, it may disappear at the aortic cartilage, and reappear with reinforcement over the great arteries of the neck, in the manner I have already described; it is lost, however, at the apex of the heart. You may find such

murmurs in cases of any profound alteration of the blood in advanced stages of tuberculosis and in carcinoma.

But, going one step farther in degree, we may find a blowing murmur heard as far as the apex, and even localized at the apex—a murmur which, from its quality and characters, is absolutely indistinguishable from that of valvular disease. The great point, however, which differentiates it from the organic murmur is its temporary or evanescent character; its occurrence corresponds with a definite period in the history of a disease, it fades away and ceases entirely when the disease enters upon a new phase.

Murmurs having these characters have been described as occurring (1) in chorea, (2) in the early stages of certain acute febrile affections.

We will consider the explanations which have been given of their occurrence—first in chorea. Every one will agree that in a certain proportion of patients suffering from chorea there is manifest a cardiac murmur, sometimes at the base, but more commonly at the apex. I have already said that this murmur is in many cases very soft, often difficult to find, and I think it is a fair inference that it is often overlooked. I know I have often overlooked it myself in the early examinations of a patient. Yet, when once it is manifested, it may be observed to be persistent in some cases, whilst in others it may completely disappear, and leave no trace of heart-affection. What is the pathology of this condition? To begin with a matter concerning which there is no doubt—in a considerable proportion of the cases of chorea which have proved fatal, vegetations have been found in greater or less degree fringing the cardiac valves.

Organic disease of the valves will undoubtedly, therefore, explain a certain proportion of the cases. Happily, however, chorea is rarely a fatal disease. Will the existence of structural changes in the valves explain the murmur which, unlike that in other valvular affections, entirely disappears? Many observers say no. How, then, do they explain the occurrence of the murmur which is heard over the mitral area? By assuming that there is in these cases a peculiar contraction of the papillary muscles of the ventricle, to which are attached the cords which control the curtains of the valve, or else a peculiar condition of the muscular walls of the ventricle, whereby the apposition of the valvular curtains is prevented, and consequently regurgitation takes place into the auricle. You will understand that this is mere hypothesis, and it seems to me very difficult to accept it. That in chorea the muscle of the heart should in some degree partake of the muscular perturbation which characterizes the disease is not impossible to realize, but whilst this would explain irregularities in the time and quality of contraction which undoubtedly do occur, it appears to me incapable of explaining the murmur. If the cardiac muscle behaved in any spasmodic manner, surely the murmur would present strange variations of site and character; sometimes it would be present and sometimes absent, sometimes soft, sometimes loud; whereas it often presents no considerable variations from hour to hour, day to day, or week to week. Spasm of the papillary muscles I cannot help but reject; disease of their muscular elements is out of the question; and any constant or consentaneous action on their part, whereby they keep open the orifice

they are intended to close, seems also to impose a too great demand upon our credulity. Parietal debility of the ventricle seems to me equally difficult to accept: it is quite unproved in these cases.

If we exclude the probability of a muscular causation, how shall we explain the non-persistent murmur of chorea? My answer is, that I see no difficulty in concluding that such murmurs are always due to some change in or on the valves. The objections taken to this view may be thus stated:—The endocarditis which produces these changes of the valves is a rheumatic endocarditis; only a small proportion of the sufferers from chorea are demonstrably rheumatic; therefore a changed condition of the heart-valves is unlikely. The second objection is the formidable one that endocardial changes of the valves do not pass away, and the murmur which they occasion is permanent. As regards the first of these objections, however, I have already shown how insidious may be the advent and course of the endocarditis, even in the form known as rheumatic, and that especially in children, the articular symptoms may be trivial or entirely absent. Moreover, there is no reason to believe that the form of endocarditis occurring in chorea is of necessity rheumatic. It is the endocarditis characterized by the presence of vegetations on the endothelial surface which is met with in chorea; this is prone to occur on valves already altered by rheumatic disease, but it can occur in the absence of rheumatism. It is a mere hyperplasia of the endothelium which may exist in very slight degree, but when it does occur the fibrine of the blood tends to adhere to the thickened spot. The obstruction caused by such a vegetation may readily occasion the mur

mur; but under the repeated washings of the current of blood, the pedicle itself and the attached fibrine may be washed away suddenly or gradually, and the valve subsequently may present no trace of lesion. I think that there is a very high probability that fragments thus derived may block some of the arterioles supplying the corpora striata or other portions of the cerebro-spinal motor tract, and that thus is produced that form of chorea which is associated with cardiac change. You must not misunderstand me, however, and conclude that in my opinion this is the *exclusive* pathogeny of chorea. It is to me highly probable that just as epilepsy is induced in some instances by actual disease of the brain and in others by reflex irritations, so chorea may be the result, in some cases of direct physical interference with the arterial supply of the motor tract, in others of the peripheral irritations (such as intestinal worms), and probably in others of psychical stimuli such as fright or emotional shock.

I shall teach you, therefore, that when in a case of chorea you hear a murmur distinctly located at apex or base you are to conclude that there is an organic lesion of the mitral or aortic valves.

We turn now to the consideration of the temporary systolic murmur which may be heard over the apex during the course of certain acute febrile affections.

A murmur with the first sound, heard over the apex of the heart but disappearing entirely with convalescence, has been observed in the course of small-pox, erysipelas, and typhoid fever. The phenomenon has been best investigated in the case of the last-mentioned disease. In certain of the cases of typhoid, about the end of the second week from the onset, a

first-sound murmur may be heard over the right ventricle or just confined to the situation of the apex. When localized at the apex the sound may have precisely the characters of an endocardial murmur, yet as convalescence approaches it entirely disappears. Certain other cardiac phenomena accompany it. The impulse of the heart may be felt to be very feeble or undulatory: the radial pulse may become intermittent; the second sound of heart may be heard to be reduplicate. The signs show great enfeeblement of the heart; this is so obvious that it does not need discussion, and it is well known that some of the subjects die suddenly in syncope.

We come to the inquiry—to what is the first-sound murmur which is heard at the apex in these cases due? We may at once exclude any organic disease of the valves, for it is known to be extremely rare and almost unexampled for endocarditis to develop in the course of typhoid, and the cases which have died in syncope have shown no trace of structural alteration of the valves. There is evidence, however, of a decided change in the muscular structure of the heart. M. Hayem, who has deeply studied this question, has found evidence of myocarditis—an inflammation of the muscular fibrillæ—with granular and fatty degeneration and a special form known as "vitreous" change. Undoubtedly, therefore, there is structural enfeeblement of the muscular walls of the heart. We have again to encounter the question: How can such enfeeblement induce the murmur? And first, is there a veritable regurgitation in such cases? M. Hayem considers that there is—the weakened muscle can but imperfectly fulfil its functions, the auriculo-ventricular orifice, powerless to resist the

force of the blood-current, allows itself to be passively distended, or else the enfeebled papillary muscles can no longer sufficiently restrain the valvular curtains. Hence a practical, though a functional, insufficiency of the valves.

The difficulty in accepting this explanation is far less than that which besets the "dynamic" hypothesis in the case of chorea. It seemed to me impossible to admit that a neurosis, the special phenomenon of which is spasm, should give rise to a long-lasting and little-varying condition of patency of the auriculo-ventricular aperture; I even found it difficult to allow that mere passive weakness of the ventricle without dilatation could permit of regurgitation. In the case of typhoid, however, we have to deal not only with general weakness but also with localized lesions. M. Hayem says that in histo-pathological examinations he has found patches of disease in the muscular fibrillæ disseminated here and there in a most irregular manner.* It is not difficult to assume, therefore, that there is a paralysis of certain of the papillary muscles, and of necessity a condition of incompetence of the valves permitting regurgitation.

Let us consider the lessons derived from a case under my own care. A young lady, aged nineteen, whom I saw on the 10th day of well-marked typhoid, manifested no cardiac murmur whatever. On the 11th day there was a very soft murmur with the first sound, localized at the third left costal cartilage. On the 13th day the bruit reached almost as far as the apex of the heart; it had all the characters of a blowing endocardial murmur; in fact, had I heard it under

* See "Le Progrès Médical," 24 Juillet, 1875, p. 416.

other circumstances, I am sure I should not have hesitated to ascribe it to valvular disease, for it was heard well within the mitral area, though careful auscultation showed that its maximum was a little right of the apex. On the 15th day it was still heard as far as the apex, but its maximum was at the third left costal cartilage. On the 17th day of the fever, the bruit was only heard in the last-mentioned situation, but there was a distinct reduplication of both the first and the second sounds of the heart. On the 21st day there was reduplication of the first sound only, the bruit still audible as before. Convalescence went on most satisfactorily, in a few days all trace of reduplication had ceased, the basic murmur passed away, and slight anæmic murmurs were manifest over the arteries of the neck. On the 38th day the patient called at my house in perfect convalescence, and presenting no signs of cardiac trouble.

This case was one of typhoid of not more than the average severity; the intestinal symptoms were well marked, and the diarrhœa continued during the second week in considerable degree; the pulmonary signs were a slight general bronchitis with condensation about the base of the left lung which soon passed through resolution; the temperature taken in the axilla never exceeded 104·2° F., nor the pulse 128, and defervescence, commencing on the nineteenth day, proceeded with almost perfect regularity. The cardiac troubles seemed to be the most grave of those which surrounded the case, and yet the course of the disease appeared in nowise to be injuriously complicated by them.

Let us review the circumstances of this case with the view of explaining the murmur. In the first

place we have positive signs of enfeeblement of the heart-muscle. A like weakness existed in marked degree in the voluntary muscles; the "subsultus" characteristic of typhoid was a prominent symptom. It is found that in these cases the changes which occur in the muscles of the heart occur also in the muscles of the body. M. Hayem concludes from his able researches that the cardiac fibrillæ are diseased in like manner and in like degree with the fibrillæ of the voluntary muscles. The existence of reduplication of *both* of the heart sounds was a very pronounced sign of cardiac implication. Let us consider the concurrent conditions. No one could doubt that in a case of this sort there was a profound deterioration of the *quality* of the blood due to the septic influences at work and to the "ensemble" of adynamic conditions. But, furthermore, there must have been a notable diminution of the normal *quantity* of the blood on which the heart could contract, for a part of its volume had drained away (and was still draining) from the alimentary canal, and another part remained stagnant in the congested lung. Such conditions were just tantamount to a notable abstraction of blood.

Cardiac debility and deterioration of blood in quality and quantity, therefore, are *positive* indications in our case. Moreover, we have signs similar to those observed in anæmia. The murmur first noticed could have been taken for an anæmic murmur localized at the commencement of the pulmonary artery. Soon it became heard over the right ventricle; in convalescence it was gradually lost, and anæmic murmurs were heard over the arteries. M. Hayem, speaking from other experiences, says of the murmur, "After having been localized just at the apex near the nipple,

it deviates to the right near the sternum, and tends little by little to ascend towards the base. At the same time the bruit becomes softer, and takes in a more and more decided manner the character of an anæmic murmur."* We have here just the factors which induce the anæmic murmurs which are heard over the aorta and the great arteries.

I have long hesitated to adopt the theory of so-called *dynamic* mitral murmurs, which I prefer to term *adynamic valvular* murmurs. As I have just said, I feel bound to reject the hypothesis in regard to the murmurs of the subjects of chorea. In the case of the mitral systolic bruit, heard in the acute fevers and in profound anæmia—a bruit which so closely resembles that which is undoubtedly due to disease of the valve, but which is nevertheless non-persistent—I am now convinced, from personal experience, that there is temporary mitral regurgitation. Dr. Hayden has published cases illustrating such murmurs in the subjects of anæmia and purpura, and of induced nervous prostration caused in one instance by habits of masturbation, and in another by excessive tobacco-smoking. In the case of the acute fevers it is not difficult to accept with M. Hayem, the view that the papillary muscles share in the disease which involves in various portions the muscular structure of the heart, that thus their co-ordinate action upon the curtains of the valve is impaired, and leakage takes place through the imperfectly closed orifice. In the cases of anæmia, &c., it is probable also that the papillary muscles are enfeebled. Guttmann says, "Inorganic murmurs at the mitral orifice are caused by the unequal tension of the segments of the valve," which "is chiefly the result of slight fatty metamorphosis of the muscular structure of the heart, more especially of the *papillary muscles*, which takes place whenever anæmia becomes profound" (*cf.* "Handbook of Physical Diagnosis," p. 287). Perl has stated that in animals fatty degeneration of the heart may be experimentally produced by repeated and copious venesection. Usually in

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* "Le Progrès Médical," 24 Juillet, 1875, p. 415.

cases presenting the adynamic murmur in the mitral area the *symptoms* of grave mitral regurgitation are absent —it is probable that the amount of blood gushing back into the auricle and occasioning the murmur is small. When the papillary muscles sufficiently recover their strength the mitral curtains become properly apposed, and the murmur disappears. Dr. Cuming, of Belfast, has described a case in which there was a mitral murmur with the usual signs of advanced organic disease--pulmonary engorgement, hæmoptysis, anasarca, &c.—and yet dissection demonstrated the absence of anomaly or disease in the cavities, walls, or valves of the heart. Dr. Hayden adds that he has also met with two such cases. In Part II. I have quoted a case of profound anæmia in which there were the usual signs of advanced organic valvular disease, and yet recovery and *total disappearance of the murmur*. In this case, relying on the evidence of the sphygmograph, I made the diagnosis that the murmur was adynamic (see Part II.). In the apex-murmurs, therefore, occurring in typhoid and other acute fevers as well as in pronounced anæmia and conditions of nervous shock, I think, with Hayem, Hayden, Guttmann, Cuming, Bamberger, Hare, and others, that there is veritable regurgitation due to enfeeblement of the muscle, chiefly of the papillary muscles, of the heart.

You will remember, therefore, whenever you hear a first-sound murmur localized at or near the apex in a case of pronounced anæmia, to consider the probability of its occurring independently of actual *disease* of the mitral valve; and, in the absence of a history of rheumatic causation, to give a hopeful prognosis, if only the condition of general anæmia can be satisfactorily recovered from. And especially if you hear such a murmur in a patient manifesting typhoid fever (if there be evidence that no mitral lesion has taken place previously to the occurrence of the fever) you may predict with the highest probability that the sound will disappear, and that the valves will not be affected.

Fugitive murmurs excluded, we now suppose that *a murmur is heard in the mitral area.* You have determined the situation of the apex-beat, and you observe that there is a bruit localized within, or having a maximum intensity within, the circumference of a circle extending an inch around the apex. The phenomenon indicates a morbid condition of the mitral orifice.

A murmur indicative of mitral lesion is met with as probably the most common of all the signs of heart-disease. Of the hundred cases of the various clinical forms of disease which I have cited, fifty-eight manifested the murmur indicating morbid change at the mitral orifice.

By far the most common cause of mitral lesions is rheumatic endocarditis. Of the fifty-eight cases just mentioned thirty-two had suffered from rheumatism, and of these twenty-three had been the subjects of rheumatic fever; two, in addition, had suffered from rheumatoid symptoms which occurred subsequently to scarlatina. In seventy cases of mitral lesions noted by Dr. Flint, rheumatism had occurred in fifty-five. You will remember that I called your attention to the frequency of pericarditis in acute rheumatism; a like frequency exists as regards endocarditis—in fact, the latter probably occurs rather more frequently than the former. Whilst Hasse, Bamberger, and Lebert give twenty-two, twenty, and seventeen per cent. respectively as the proportion of cases of endocarditis occurring in the course of rheumatic fever, Fuller and others have fixed the percentage much higher. Not at all infrequently the two diseases, endocarditis and pericarditis, occur together during acute rheumatism. I have already hinted that endocarditis, like pericarditis, can arise and run its course in subacute rheu-

matism, where the articular symptoms are very slight indeed—in fact, that in some cases objective symptoms may be entirely absent. We must conclude, therefore, that endocarditis of the rheumatic form may occasionally occur very insidiously, so that its origin and course may be entirely overlooked.

Rheumatic endocarditis commonly starts from the mitral valve; in many cases the disease does not extend further. It is an obvious corollary that the mitral is the most frequent site of valvular deterioration. Combining the figures of Willigk, Flint, and Cockle, it would appear that the mitral is affected in one hundred and sixty-six cases to one hundred and thirty in which the aortic valves are diseased. The disease spreads from the mitral valve to the endocardium lining auricle and ventricle; its effects may often be traced by the appearance of a milky patch of thickened endocardium, stretching in a direct path across the ventricle from the mitral to the aortic valves. The pathological changes occurring in rheumatic endocarditis consist, first, in a swelling and thickening of the serous membrane and the substance of the valve, the microscope showing an increase in the number of connective tissue nuclei; subsequently there is much development of fibrous tissue; lastly, there is a gradual process of retraction of the newly-formed tissue just as occurs in cicatrices. The morbid process may involve the muscular structures immediately subjacent—there may be myocarditis, with the result of shortening of the papillary muscles and consequent retraction of the cords and curtains of the valve.

Other causes besides rheumatic endocarditis may produce lesion of the mitral valve and orifice. These are identical with those which we have already con-

sidered as affecting the aortic valves—atheroma and endocarditis of the villous (*i.e.*, accompanied by vegetations) and the ulcerative forms. The valve may be incrusted by calcareous salts, and rendered hard as bone. Endocarditis may be induced by renal disease. Lancereaux has recorded a very interesting case, in which, with a condition of contracted kidney, there was disease of the mitral valve characterized by thickening and the appearance of vegetations which, under the microscope, were seen to be studded with granules, distinctly proved to be deposits of *urates*. The probabilities are very great that rheumatic endocarditis is due to the presence or excess in the blood of the acid products of tissue disintegration, and that the endocarditis met with in renal disease is due to retention of the products which the kidneys are unable to excrete. In the villous form of endocarditis little excrescences may be seen fringing the margin of the mitral orifice, especially on the auricular side; these have been often observed after death in cases of chorea. The indications of ulcerative endocarditis we have briefly considered in connection with the aortic valves; remember that it may in like manner attack the mitral: recent observations have shown that it may occur as one of the sequelæ of parturition. Another cause of lesion of the mitral orifice is rupture of the tendinous cords connecting the curtains of the valve with the papillary muscles. This is not very uncommon; Flint found it in four out of thirty-nine cases of mitral defect; it may occur from violent action of the heart. Again, the mitral orifice may be rendered imperfect on account of dilatation of the ventricle, whereby approximation of the curtains of the valves is prevented. Lastly, but

very rarely, fusion together, or perforations, of the mitral valve may be congenital.

When we come to review these various pathological causes in relation to their effect upon the auriculo-ventricular orifice, we find that they can produce two well-marked varieties of lesion, as well as a third variety, which is a compound of both the others. Thus the orifice may be *patent*, the valve imperfectly closing it; or it may be *obstructed*, the outlet from the auricle being narrowed; or it may be both *obstructed and patent*, an impediment existing to the outflow from the auricle, as well as an imperfection of the valve whereby it is prevented from closing the orifice.

The first of these conditions is the most common. By thickening and shrinking of the curtains of the valve, by the presence of vegetations distorting them or weighing them down, by their "pouching" with aneurismal dilatations, by their perforation or ulceration, by swelling and shortening of the tendinous cords attached to the curtains, by deformity or rupture of the musculi papillares, by disease and retraction of the muscular wall of the ventricle, or by such dilatation of it that the edges of the valves cannot meet—by these and some other causes the mitral orifice is prevented from closure at the time of the systole of the heart. There is then said to be a condition of *mitral insufficiency;* that is, the valve is insufficient to close the auriculo-ventricular aperture. The consequence is that at each contraction of the ventricle a portion of the content of blood gushes backwards, through the space left by the imperfect apposition of the valve, into the left auricle. There is said to be *mitral regurgitation.* One consequence of

this condition is that the auricle is always abnormally full; the continuance of this leads to *dilatation of the auricle*, and the amount of such dilatation affords an index of the amount of regurgitation. Furthermore, the ventricle is of necessity incompletely emptied; from containing habitually too much blood it also becomes *dilated*, and, in obedience to the law which enables involuntary muscle to increase in bulk and strength in order to overcome obstacles, it becomes *hypertrophied*. This condition of hypertrophy with dilatation has been called eccentric hypertrophy. The consequences on the general system arising from the leakage through the mitral orifice are, unless the condition of hypertrophy exactly compensate for the evils, insufficient supply of blood through the aorta, whence diminished blood-pressure in the arteries, sluggishness of flow in the capillaries from impaired *vis à tergo*, and undue repletion of the venous radicles and the general venous system. And when we come to the right heart we find a condition of distension (the pulmonary second sound is intensified, owing to the heightened blood-pressure, as I have before pointed out), and thus the right cavities may become dilated. In the majority of cases the cause inducing the condition of mitral regurgitation is *rheumatic endocarditis*. Mitral regurgitation occurred in forty-eight out of my hundred cases of heart disease. Of these twenty-six had suffered rheumatism, nineteen having had rheumatic fever; besides, two had suffered scarlatina, wherein you know there are rheumatoid phenomena. Chorea existed in two cases, gout occurred in one.

A quite different set of conditions obtains when the mitral orifice is obstructed. The average circumference

of the normal orifice, according to Bizot, is four inches; its form is oval, the long diameter being one inch. These dimensions are greatly modified by disease. The cords, curtains, and muscles of the valve may be stiffened into a rigid mass, or the edges of the orifice may be obstructed by vegetations, in some cases very small and insignificant, in others assuming the dimensions of large polypi. In many cases the form of the valve curtains and the shape of the outlet are singularly altered. The curtains are, as it were, fused together into an even tube, more or less conical, with its smaller extremity downwards opening into the ventricle: sometimes this extremity is circular and may be extremely small; it may admit only the thumb or the little finger, or be so minute as scarcely to allow a small catheter or even a crow's quill to pass through it. This variety is known as the "funnel-mitral." Or the free extremity, instead of being circular, may be slit-like. Dr. Hilton Fagge has recorded a case in which the slit was so narrow that it would not admit a three-pennypiece edgewise. This is called the "button-hole" mitral. The septum formed by the adherent valve-curtains may be incrusted with calcareous salts so as to be of bony hardness. In many cases, however, it is perfectly smooth and so uniform in its conformation as a hollow cone, that it seems to suggest that it must be a congenital anomaly; its perfect regularity appearing to contradict the probability that it is the product of disease.

The effect of these conditions upon the auriculo-ventricular aperture is the reverse of that produced by the conditions which permit regurgitation. The aperture guarded by the mitral valve, instead of being widened, is narrowed—there is said to be *mitral stenosis.*

There exists an impediment to the flow from the auricle into the ventricle—there is *mitral obstruction*. Such an occurrence is not uncommon. Flint found stenosis in sixteen cases out of thirty-nine instances of mitral lesion. Of my forty-eight cases of mitral disease, stenosis was declared by the physical signs to exist in ten.

As I have before said, the regularity of form in the case of the fused mitral valve has suggested the probability of a congenital causation. Dr. Hilton Fagge inclines to this view in some cases, and thinks that a rheumatic origin is comparatively rare. For my own part, however, I am disposed to the view that nearly all the cases are the result of rheumatic endocarditis of a very chronic form. Of the ten cases I have mentioned, four had suffered from rheumatic fever, and two from subacute rheumatism; in the remaining four the causation was hypothetical. In undoubtedly rheumatic cases, however, I have seen the funnel mitral of the precisely regular conformation suggestive of the congenital anomaly, and I think it must be explained by the involvement of the whole texture of the valve by one or successive attacks of endocarditis (which attacks, as I have before said, may occur with little or no subjective sign), and by slow and regular development of fibrous tissue with quasi-cicatricial change. The great rarity of the positive evidence of congenital anomaly of the mitral valve is in favour of this view.

The secondary effects of mitral obstruction are quite different from those of mitral regurgitation. The most constant and most pronounced effect is upon the left auricle, the wall of which becomes *hypertrophied*.

I have already cited a case under my own care illustrative of this point in considering the chronometry of pulsations. It is quite clear that the hypertrophy occurs in obedience to the usual rule—there is an impediment to the outflow from the auricle to the ventricle, and the muscle of the auricle becomes hypertrophied to overcome the obstruction. Whilst dilatation of the left auricle without hypertrophy is characteristic of mitral regurgitation, dilatation *with* hypertrophy is characteristic of mitral obstruction. The left ventricle in mitral obstruction is found not to be dilated. In some cases it has been observed to be smaller than normal, its muscular wall has rarely been found hypertrophied. When it has been thus found the condition has been that formerly known as *concentric* hypertrophy, which simply means hypertrophy without dilatation. In the great majority of cases the left ventricle in mitral obstruction is found not obviously abnormal, the condition thus differing from that in mitral regurgitation wherein dilatation and hypertrophy are the rule.

The consecutive changes in the right heart are alike in regurgitation and obstruction, though the initial causes are different. In both conditions there is impaired *vis à tergo*, and hence venous congestion and dilatation of the cavities of the right heart, but this is due in the case of stenosis to the imperfection of supply from the auricle to the ventricle with retention in the auricle, whilst in regurgitation it is due to the retrograde diversion of the blood-stream through the mitral aperture.

The subjoined diagrammatic sketches may aid you to comprehend the conditions in mitral stenosis, as

distinguished from those in mitral regurgitation. They are intended to represent vertical sections through the left auricle and ventricle.

FIG. 4.

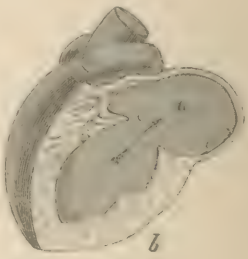
MITRAL STENOSIS (Obstruction).

a Hypertrophied muscular wall of left auricle.

. Section of wall of left ventricle.

c, Narrowed auriculo-ventricular orifice.

FIG. 5.



MITRAL INSUFFICIENCY (Regurgitation).

a, Dilated left auricle

b. Section of hypertrophied and dilated left ventricle.

c, Patent auriculo-ventricular orifice.

(The arrow shows the direction of the current of blood at the time of the production of the murmur.)

LECTURE VIII.

AUSCULTATION.

PART IV.

Differential diagnosis of mitral lesions—Rhythm—Chronometry of murmurs—Vocal representation of murmurs—Graphic representation of murmurs—Area of audibility—Signs of mitral stenosis—Signs of mitral insufficiency—Double mitral murmur—Combined murmurs—Diagnosis of complex lesions.

SEEING that there are these well-marked varieties of lesion of the mitral orifice, the question now occurs—can they be detected and differentiated during life? The answer is undoubtedly in the affirmative.

The previous examination of the patient has given many data. We must exclude the evidence of any departure from the normal as regards the right chambers of the heart, for consecutive changes in these can occur from either of the abnormal conditions of the mitral orifice. Our examination of the left chambers, however, may have afforded valuable evidence. If we have found from the physical signs that the left ventricle is dilated or dilated and hypertrophied, the probability is great that if there be any mitral lesion at all it will be one of *regurgitation*. Palpation may strengthen this probability by evidencing a systolic thrill; remember, however, to time it carefully, for systolic thrill is rare and presystolic thrill by far the more common. Suppos-

ing, on the other hand, that we find little or no evidence of displacement of the apex, but a thrill communicated to the finger distinctly antecedently to the impulse of the heart—in this case the diagnosis of mitral *obstruction* may be positively made. In some such cases as I have before shown, the pulsation of the hypertrophied left auricle may be visible in vibration of the chest-wall, and its movement immediately before the ventricular systole can be demonstrated.

It is but a small proportion only, however, of the cases of obstruction of the mitral orifice that can be thus diagnosed. The question now becomes narrower: can the conditions be differentiated by auscultation? Twenty years ago it would have been declared impossible. Contraction of the mitral orifice was revealed by post-mortem examinations in a large number of cases then as now. The diseased condition of the valve was not overlooked, but obstructive and regurgitant lesions were no doubt mingled together in clinical diagnosis. A murmur was heard in both classes of cases, but the rhythm of such murmur was not recognized. Although some steps had been taken in this direction by French observers, Fauvel and others, it was not till Dr. W. T. Gairdner, in 1861, by careful clinical observation and philosophical demonstration showed the characters, the import and the causation of the murmur dependent upon mitral constriction, that the diagnosis in any considerable number of cases was affected. Further observations, especially those of the late Dr. Hyde Salter and Dr. Hilton Fagge, have contributed to spread the knowlege of the methods of discriminating the two conditions, but there is not the least doubt

that even now the condition of mitral stenosis in a large number of cases is not differentiated from the regurgitant lesion. As to the ease with which this diagnosis can be effected the opinions of good observers vary; some say, with Dr. Hyde Salter, that any one who should fail to recognize the murmur of mitral obstruction "could hardly be considered a decently informed member of our profession;" others, with abundant opportunities of observation, have failed to discover the murmur, and have written papers to prove that it has no existence. The truth lies probably between these two extremes. The typical presystolic murmur any instructed clinical observer who takes the necessary time and trouble cannot fail to recognize, but there are some murmurs so short, so slight, and so obscure that their recognition and the determination of their rhythm are matters of very great difficulty. Such obscurities are, however, quite exceptional, and in the great majority of cases it will be your own fault if you fail to diagnose mitral stenosis when it exists.

We must first inquire: what is the rhythm of the murmurs heard at the apex of the heart? At the base we know that the rhythm is very simple. Of the murmurs there localized one is heard with the first sound (systolic), the other commences with the second sound (diastolic). At the apex we may have, as at the base, a murmur coincident with the first sound—systolic murmurs may occur both at the base and apex—but, as a matter of fact, a murmur whose origin is at the mitral orifice scarcely ever commences at the same period of the heart's rhythm as the diastolic murmur heard at the base. Such mitral murmur occurs not *with* the second sound, but *after*

it. Careful observation will show that a mitral murmur is not diastolic in the sense of being absolutely coincident with the diastolic or second sound of the heart, but that it occurs *after* the second sound and *before* the first sound. It is now well known as the presystolic murmur. A strictly diastolic murmur of mitral origin—that is, a murmur commencing at the time of the second sound with a pause before the first sound—although extremely rare, is not unknown. Its mechanism will be considered hereafter. It is unfortunate that the German authorities describe all those murmurs generated at the mitral orifice which are not systolic as *diastolic*. Under this term they include the presystolic murmur—in fact, all sounds that occur between the second sound and the succeeding first sound. (*Cf.* Guttmann, "Handbook of Physical Diagnosis," p. 289.) I have already described to you the mechanism of presystolic thrill and presystolic pulsation. The murmur which we are now considering is produced by the same cause—the forcing of the blood through the narrowed auriculo-ventricular orifice.

The term *presystolic* is not wholly devoid of objection. It is obvious that the systole of the heart means the systole both of auricles and ventricles—the presystolic murmur is presystolic only as regards the ventricular systole, it is coincident with the auricular systole. Nevertheless, the term has been so useful that we cannot wholly discard it. Dr. Gairdner proposed the term *auricular-systolic*, and this is for the most part expressive of its physiological causation. The propriety of the terms will, I consider, necessarily vary as these are employed for the expression of the *clinical* or the *pathological* conditions. For the latter

purpose, Dr. Gairdner's term is usually appropriate: but for clinical record it is better, in my opinion, to use a term which shall fix the period of the murmur in the heart's rhythm without even the semblance of hypothesis. I do not think that the use of the words "systolic" and "diastolic," for the purpose of timing murmurs, is without reproach: but these, as well as "presystolic," have become incorporate with our notions, and we cannot well do without them. We use them, however, on the principle expressed by the phrase: If you know what I mean, what does it matter what I say? For precision, it would be much better, in my humble opinion, if heart murmurs were expressed in plain English and indicated by the periods of the obvious and precise sounds of the normal heart. Thus, a systolic should be expressed as a *first-sound* murmur, a diastolic as a *second-sound* murmur, a presystolic as a *before-first-sound* murmur.

From this preface let us turn to the practical methods of discriminating the rhythm of the murmurs heard over the mitral area and dependent upon disease of the mitral valve. Here let me say, that you must give full and long attention; for the diagnosis is easy if you take sufficient care, confused and obscure otherwise.

First.—Endeavour to determine the exact position of the murmur in the heart's rhythm. We may call this the method of CHRONOMETRY OF MURMURS. The principles of it I have already applied in the cases of pulsations occurring over the cardiac area and the phenomena of thrill. At the risk of frequent repetition I will again state the rule:—Apply your stethoscope over the point of maximum loudness of the murmur, and place the tips of the fingers on any

point of the chest where you can feel the impulse of the ventricles or over the site of the pulse of the carotids. You auscultate at the same time that you feel the pulsation of the heart or of one of the great arteries near it. The question which you have to ask yourself is this: Is the murmur which I hear coincident with the first sound of the heart, or is it previous to it and directly terminated by it? Do not run away with the notion that without all this care you can determine the question: you may not be able even to distinguish the first sound from the second sound. What said one of the greatest authorities, the late Dr. Stokes?—"So great is the difficulty, that we cannot resist altering our opinions from day to day as to which is the first and which is the second sound."* There can, I think, be no doubt that the great reasons why in times past the condition of mitral stenosis remained undistinguished from that of regurgitation, was that the murmur was judged to be systolic, and the first sound was taken for the second sound. In cases of stenosis, the mistake is peculiarly easy, for the first sound is usually very short, sharp, and sudden, much resembling the second sound. These considerations are quite enough to inculcate lessons of care in observation, and the absolute necessity of timing the murmurs. If the murmur be systolic you will hear it commence at the same instant that the finger is sensible of the cardiac pulsation. It is produced, as you know, by the reflux current forced into the left auricle by the ventricular contraction. It is a truism, therefore, to say that its period of production is the period of its cause—*i.e.*, the

* "A Practical Treatise on Diseases of the Heart," 3rd edition, 1862, quoted by Dr. Fagge, *loc. cit.*

ventricular systole. Commencing immediately with the full force of the contraction, it may last throughout the whole systole, or it may become feebler and inaudible as the ventricle becomes emptied. In other words, it may be short or long; commencing with the systole, its duration may be the whole or a part thereof. Such is the first-sound murmur,—the murmur of mitral regurgitation.

Suppose now that the murmur which you hear be not coincident with the systole. At the moment that your finger becomes sensible of the impulse of the heart against the wall of the chest, the flap or thud of the first sound is audible. Preceding this, however, and soon after the second sound, a rough murmur is heard. This murmur also may be either short or long; it may commence immediately after the second sound, or it may occur momentarily before the first sound, but the latter always terminates it as with a sudden full stop. The mechanism I have before explained—it is the sound produced by the effort of a hypertrophied auricle in urging the blood through a narrowed mitral orifice. This presystolic, before-first-sound murmur, is absolutely diagnostic of mitral stenosis.

Secondly.—In order to help you to realize the distinctive characters of these sounds, I would call your attention to a method of Vocal Representation of Murmurs. The "bruits" heard over the heart-region have received since the time of Laennec many names according to their nature and quality. Thus we have bellows' sounds, filing, grating, rasping, croaking, crowing, whining, caterwauling, and blubbering sounds, or musical or sibilant sounds. Bouillaud imitated the character and pitch of the sounds

by letters, the pronunciation of S expressing the extreme of sibilant murmurs of high pitch, R representing those of low pitch. These illustrations are all of some value, as enabling one to note the variations in character of any given murmur from time to time, and thus indicating changes favourable or unfavourable, or the persistency of the lesion. Any of the illustrations just given may be applied to the murmurs produced by the valvular conditions we have as yet considered; the sounds, therefore, cannot be considered diagnostic. Valuable help, however, may be derived from a consideration of the sound in the differential diagnosis of murmurs heard over the mitral area. The murmur of regurgitation may have almost any of the characters just described, but not so the murmur of obstruction. This latter is almost invariably rough; it has been called a "churning," "grinding," or "blubbering" murmur. In my own opinion, in typical cases, it may be best illustrated as "rolling," or "bubbling," resembling the sound of air rising through water. You may easily realize its distinctiveness from the systolic murmur by these simple considerations—the systolic fades off but never terminates abruptly, the presystolic always stops suddenly. I have frequently illustrated this by the subjoined vocal illustration, the sound of "*p*" indicating the abrupt termination of the presystolic murmur.

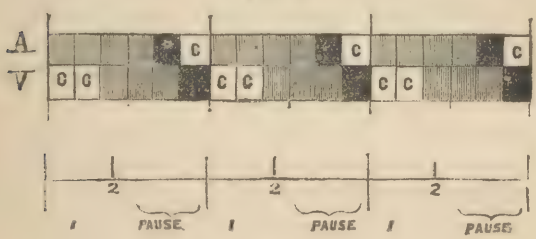
Sound of Systolic Murmur.	Sound of Presystolic Murmur.
Hook—hoof—ruff.	*Up—rup—rr. rr. rup.*

Thirdly.—We come to a plan which we owe to Dr. Gairdner, and I know of none which is more valuable to fix upon the mind the phenomena of the

murmur, and to serve as a method of record. We may term this the method of GRAPHIC REPRESENTATION OF MURMURS. To represent all the facts and conditions, I have combined Dr. Gairdner's plan with a chart which I have modified from Dr. Salter.

The upper portion of this diagram represents the conditions of the auricle and ventricle during three cardiac pulsations. Each such pulsation or cycle is divided into six equal parts, which are denoted by the squares. The squares which are shaded denote that the chamber (auricle or ventricle) contains blood, whilst the black squares indicate that it is replete—*i.e.*, at its maximum of distension. The squares marked C denote contraction or systole. The upper line of squares A pertain to the auricle, the lower V to the ventricle. Taking the auricle first and proceeding from left to right, we see that it is receiving

FIG. 6.



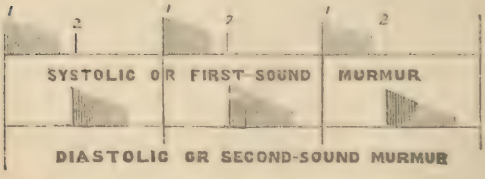
THE NORMAL CARDIAC RHYTHM.

blood during the period indicated by the first four squares, and that at the fifth it has arrived at its maximum distension. Then follows C, its systole or emptying through muscular contraction. Looking at the ventricular line V, we see that the systole lasts during the first two squares, or two-sixths of the

cycle, that during the following three it is in diastole or receiving blood, and at the sixth it has arrived at its maximum distension. Then the cycle is in each case repeated. Now, we are enabled by a glance at the diagram to note the relative condition of auricle and ventricle at any given moment of the heart's action; we have only to compare the upper sections with the lower. Thus we see that whilst the ventricle V is in systole, the auricle A is in diastole: that A, having at last become distended, contracts, while V is yet in diastole, and completes the repletion of V, which recommences the cycle by contraction. Again, by a horizontal line below, we have a means of comparing these conditions with the sounds of the normal heart, the vertical lines 1 and 2 denoting the first and the second sounds respectively. You will see that the latter occurs very soon (about half a square) after the ventricular contraction, the first sound lasting during two squares.

We have thus a very simple and concise method of registering murmurs, their position in the cardiac

Fig. 7.



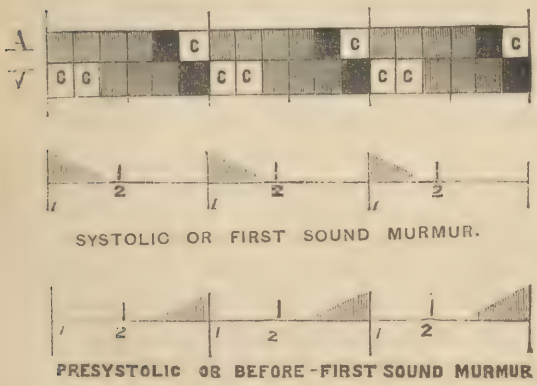
rhythm and their duration. Taking the horizontal line to express the duration of the cycle, and the vertical lines to denote the first and second sounds, we have merely to indicate by shading the place and

estimated length of the murmur. Thus, the simple systolic and diastolic murmurs heard at the *base of the heart*, would be indicated by the diagram (fig. 7). The one commences with the first, the other with the second sound.

The rhythm of the first-sound murmur heard at base and apex is precisely similar. The upper line of this diagram would alike express the sounds of aortic obstruction and mitral regurgitation. Of course a note of the area of audibility and maximum intensity would at once indicate the diagnosis.

We will now consider how this method illustrates the differential diagnosis between the murmurs of mitral regurgitation and mitral stenosis. The subjoined diagram you will readily understand from the

Fig. 8.



explanation I have already given. It shows how the *systolic* murmur starts *with* the first sound, and the *presystolic* leads up *to* the first sound; whilst a glance

at the upper section of the diagram indicates the cardiac conditions at the time of the production of the murmurs. The first-sound murmur occurs with V C C, the ventricular contraction; the presystolic with A C, the auricular contraction. It is possible that, as the diagram shows, the presystolic murmur may commence during the distension of the auricle, and previously to its actual systole.

Having considered the *rhythm* of the murmur, we have yet another aid towards the differentiation of mitral stenosis from mitral regurgitation in the determination of the *area of audibility*.

It is rare for the murmur of stenosis to be heard to any considerable degree below or outside the situation of the normal apex. As I have before said, the conditions of hypertrophy and dilatation of the left ventricle, which cause the apex-beat to be discernible externally and inferiorly to the normal position, seldom coexist with mitral stenosis. A murmur, therefore, heard left of the cardiac area in conjunction with the signs of ventricular enlargement is unlikely to be a murmur of mitral obstruction. Exceptions to this rule occur when hypertrophy of the ventricle from other causes coexists with mitral stenosis. I have recorded a case in point in which there had been repeated attacks of pericarditis with numerous adhesions.* Observers have agreed that the murmur of obstruction is a localized murmur—it is seldom audible far from the apex. Again it is said to have its maximum intensity *at* the apex. To this last conclusion, however, I shall have to demur: it has been an almost invariable experience with me

* Vide *Medical Times and Gazette*, June 10th, 1874, p. 34.

that the presystolic murmur is loudest at a point internal to (*i.e.*, right of) the apex-beat. It is usually audible over a portion of the normal area of the right ventricle, but seldom as far on the pulmonary cartilage, but is suddenly cut off at the left apex and is inaudible, or scarcely audible, left of the apex.

Let us contrast this with the murmur of mitral regurgitation. This systolic murmur may have a very small area of audibility around the apex, but its maximum is *never* just internal to this point. On the contrary, in a large majority of cases it is louder a short distance outside the apex than at the apex itself. When it is of considerable intensity, the sound of the murmur may be carried towards the axilla and may be audible over the axillary part of the chest-wall: it will probably disappear as the stethoscope is carried to the back, but may again become evident between the scapulæ, especially in the interval between the angle and the spine of the left scapula.

In exceptional cases both the systolic and the presystolic murmurs, generated at the mitral orifice, may be conducted towards the *right* instead of to the left apex. In these there is an unusual conduction of the murmur, due, according to my observation and experience, to the fact that the disease of the valve has involved that portion whose papillary muscles and cords are connected with the inter-ventricular septum. So the vibrations are conducted by the dense material through the septum to the sternum and the right apex, and the maximum of the murmur is in the tricuspid area. In like manner a tactile thrill of systolic rhythm may be abnormally conducted towards the right. I have also found the presystolic murmur under like circumstances conducted to the tricuspid region. Where the bruit is loud and musical or whistling, I consider that we may infer that the diseased portions of the valve have undergone calcareous transformation. Guided by this experience, I made, in a case which presented a presystolic murmur having these characters (but which ceased at the later stages of the disease), as well as a

systolic mitral murmur, the following diagnosis:—That there was mitral constriction, the characteristic presystolic murmur of which had been conducted by dense material to the interventricular septum and thence towards the right apex; that the valve-aperture was probably a calcareous ring; that the murmur had ceased to be audible because the left auricle had become dilated and its muscle enfeebled; that there was, in addition, incompetency both of mitral and tricuspid valves, with hypertrophy and dilatation of each ventricle. At the autopsy, in which I was kindly assisted by Dr. Hamilton of Canonbury, who had had daily charge of the patient, the diagnosis was exactly confirmed. The left auricle was very large and its walls extremely thin. Seen from the auricular aspect, the mitral orifice was of button-hole shape, with rigid borders, and allowing abundant regurgitation; the right boundary was converted into a dense calcareous mass with numerous hard and rough projections. The tricuspid orifice was moderately incompetent, but its valves normal. It thus appears that in certain cases of murmurs heard in the *tricuspid* area we may be able not only to infer that the morbid change is at the *mitral* orifice, but to diagnose the nature of the deposit. (Cf. "Proceedings of the Medical Society of London," vol. iii., p. 145, and *Medical Examiner*, Jan. 25, 1877, p. 65.) Guttmann, following Naunyn, says that in very exceptional cases a mitral systolic murmur is of greatest intensity in the *second left intercostal space*. Naunyn believes this to indicate hypertrophy of the left auricular appendix:—"That, as in every case of mitral insufficiency the systolic regurgitant current of blood, rushing from the left ventricle, enters not only the corresponding auricle, but penetrates also to its appendix (the cavities of both parts being continuous), the further the latter passes round the pulmonary artery, and the nearer its apex comes to the anterior chest-wall, the more favourable are the conditions presented for the propagation of the mitral murmur through the left auricle into the appendix and thence to the thoracic parietes (Guttmann, "Hand-book of Physical Diagnosis," Sydenham Society's translation, p. 289). This seems to me a far-fetched theory: it is far more likely that the murmur, as in the instances I have quoted, is abnormally conducted by the diseased structures at the insertion of the right cuspid of the mitral valve.

A little consideration will, I think, enable you to realize the physiological causes of the differences in site of the two murmurs. A reference to the diagram illustrating the conditions in *mitral stenosis* (p. 188) will remind you that the direction of the current—*i.e.*, the line of convection of the murmur—is from the auricle *to* the apex of the heart. It would seem, therefore, *primâ facie* that the "bruit" should be most audible *at* the apex. We must recollect, however, that at the time of production of the murmur the apex is not close to the wall of the chest; its position is slightly internal to that which it occupies when the ventricle strikes the chest-wall, and a space intervenes. These considerations to my mind explain the position and the limitation of the murmur. In *regurgitation*, on the other hand (see fig. 5), the direction of convection is contrariwise; you might say that the sound ought to be carried to the region of the left auricle. Why is it not so? Surely because the walls of the left ventricle conduct the sound in a direct line to the apex, and thence to the ear. At the moment of production of the first sound the apex is in direct contact with the chest-wall, and occupies a position left of that which it occupied previously. The muscular ventricle, the thoracic parietes and the stethoscope, being in close apposition, constitute one solid conductor through which the sonorous vibrations travel in uninterrupted course to the ear. Both these murmurs may be intensified by causing the patient to use muscular exertion; but especially the presystolic. The latter is apt to vary in intensity and audibility: it may be absolutely inaudible till the patient is put through a little walking exercise.

We will now sum up our auscultatory evidence.

A rough murmur is heard antecedently to the first sound, and abruptly terminated by it: its maximum intensity being slightly internal to the normal apex. The condition is one of MITRAL STENOSIS.

We will devote a short time to the clinical history of these cases, and the phenomena which you may observe during your observation of their progress. The late Dr. Hyde Salter noticed the remarkable proclivity of the subjects of mitral stenosis to *hæmoptysis.* He recorded this as occurring in six out of eight cases. I have no doubt, however, that this was an exceptional frequency; for out of sixteen cases recorded by Dr. Fagge, I find hæmoptysis noted only in three, and in seventeen cases under my own care it occurred also in three. Though these figures modify those of Dr. Salter, they support his conclusion that hæmoptysis is a frequent symptom in mitral obstruction. The cause is no doubt the backward pressure exerted by the contraction of the auricle (opposed as it is by the obstruction at the auriculo-ventricular outlet) upon the pulmonary veins; hence there is congestion of the pulmonary capillaries to the point of rupture. In mitral obstruction the capillaries suffer the *direct* pressure of the contracting auricle, whilst in regurgitation the auricle intervenes as a dilatable cavity between the contracting ventricle and the pulmonary veins.

The next point of interest in the clinical history of these cases is the proneness to embolism of one of the cerebral arteries, or of some artery of the lower limb. Remember, therefore, to inquire carefully into the cerebral conditions of all patients whom you find to manifest the signs of mitral stenosis, and conversely in cases of *sudden* paralysis, apparently of cerebral

origin, be mindful to explore the mitral region. You will readily understand how the contraction of the auricle may detach a pellet of fibrine or a pediculated vegetation from the endocardial surface, or from the margin of the auriculo-ventricular orifice, and transmit it into the direct current of blood urged by the ventricle into the arteries.

With regard to other symptoms, the subjects of stenosis are in most respects affected in like manner with the subjects of insufficiency. As I have said before, the secondary effects upon the right chambers of the heart are alike in both cases. So we have in both cough, dyspnœa, and the evidences of congestion, pulmonary and general, and in the end, dropsy, &c. In one point, in my experience, the cases are slightly different—the subjects of stenosis are more liable to variable symptoms and spasmodic troubles.

Mitral stenosis excluded, we turn to the other condition.

A murmur is heard with the first sound, or entirely occupying the place of the first sound in the cardiac rhythm: its maximum, at, or external to, the position of the apex-beat. The condition is that of MITRAL INSUFFICIENCY.

In these cases of mitral regurgitation, when the lesion is not compensated by exactly sufficient hypertrophy of the ventricle, the symptoms may be any of those which we have already considered early in these lectures. It is unnecessary to revert to them.

With regard to *Prognosis* in stenosis and insufficiency respectively, opinions are divided. For my own part, whilst agreeing that in stenosis there are certain special dangers, I am inclined to the belief that on the whole compensation is more certain and more persistent.

We come now to consider the case wherein the conditions we have just discussed coexist.

A murmur is heard antecedently to the first sound, and in addition a murmur is heard supplanting, or occurring with, the first sound. The condition is one of combined MITRAL OBSTRUCTION and MITRAL REGURGITATION.

In a very small minority of cases the sound is distinctly double. At the situation of the apex-beat you hear a murmur with the systole, then a pause, then a murmur which seems to be diastolic, and then a short pause before the recurrence of the first-sound murmur. You can convince yourself, by carefully timing, that the murmur which appears to be diastolic is really subsequent to the click of the semilunar valves. I am aware that in saying that there may be a pause between a presystolic and a systolic murmur, I am diverging from the teaching of some who have deeply studied the question, but I do not speak without practical experience. I had an opportunity of observing a case in which this was exemplified. At a spot just internal to the apex there were two distinct murmurs separated by pauses. The explanation of the phenomenon appeared to be given to another case under my care, in which the only endocardial murmur was presystolic, but this was separated by a decided pause from the first sound. It was of such character that I called it a diastolic murmur. In this case the post-mortem examination revealed extreme narrowing of the mitral orifice, but in addition the ventricle was, in great measure, filled by vegetations depending from the lower surface of the valve. The latter condition seemed to me to explain the peculiarity of the murmur —the ventricle being already partially filled by the

vegetations, the murmur occurred only at the early part of the diastole, ceasing early because the small ventricular space left became quickly replete.

In the great majority of cases the double mitral murmur can be readily resolved into two murmurs, having the distinctive characters which we have already discussed. The systolic element of the compound murmur is widely diffused, but the presystolic is heard only over its usual limited area. In the near neighbourhood of the apex the sound may be distinctly double: you hear the rolling presystolic murmur pass into the prolonged systolic. Or the presystolic may abruptly cease with a sharp first sound from which a soft systolic murmur tails off. In many cases, however, there is no spot where you can hear the two murmurs at one and the same time. This was well described by the late Dr. Salter, who cited a case. In Dr. Salter's words—"At the apex a grinding bruit is heard, *immediately preceding an apparently natural first sound*, and terminated by it. On working back well into the axilla, and not until the axilla is quite reached, a *systolic* murmur begins to reveal itself, increasing in distinctness as you work round to the back, where it is loud and strong. It is audible over the whole of the left side of the back. On returning again to the front, to the region of the apex, it is quite lost, and the presystolic bruit is again heard."* I have no doubt that you will meet with examples verifying this description.

The physical cause of mitral murmurs can be explained according to the same principles as those which govern murmurs generated at the aortic orifice.

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* *Lancet*, July 24th, 1869, p. 114.

As regards the mitral *systolic* murmur we can thus explain its production:—So long as at the time of systole the curtains of the mitral valve are perfectly apposed there results only the sound of valve-tension—the normal first sound. Vibration is prevented by the coaptation of the valve-curtains. If from any cause, however, the segments do not meet, their borders are free to vibrate in the stream of blood, which of necessity regurgitates through the orifice left by their imperfect approximation. Where the gap is considerable, or the disorganization of the valve extensive, the normal sound of valve tension is altogether lost by the murmur entirely *replacing* it. When, however, a considerable portion of the valve curtains are capable of flapping back, the murmur is affixed to the sound of their closure. Such murmur is sometimes termed *post-systolic*. It must not be forgotten, however, that the sound of valve tension may be that of the tricuspid; if the murmur, when heard at the axilla and back, is accompanied by the flap of the valve, it may be fairly inferred that a considerable portion of the mitral valve is competent. The vibrations communicated to the boundaries of the imperfectly closed orifice are not conveyed by the current, as in the case of the aortic regurgitant murmur, for in such case the "bruit" would be heard over the left auricle. They are, however, conducted by the solid structures, usually to the wall of the ventricle and the left apex, exceptionally, as lately explained, by good conductors, to the right apex and the adjacent sternum. The mechanism of the mitral systolic murmur is simple, and its occurrence pathognomonic of mitral regurgitation. The loudness of the murmur is by no means an index of the amount of regurgitation. In

the earlier stages of valvular insufficiency it may be almost confidently stated that there can be no regurgitation without murmur. When, however, as in the last stages, the left ventricle becomes very feeble, the murmur may cease to be generated. So the enfeeblement or cessation of a mitral systolic murmur, when there are signs of increasing cardiac distress, is an evil omen pointing to failure of the left ventricle. On the other hand, when the disappearance of a mitral murmur is coexistent with amelioration in cardiac symptoms, and especially with enhanced power of the left ventricle, as evidenced by the cardiograph and sphygmograph, the prognosis is good; the regurgitation has been probably due to muscular enfeeblement, and not to structural disease of the valve.

The *presystolic* mitral murmur I consider to be due to vibrations communicated to the abnormal mitral curtains, the solid structures in union with them, and the wall of the left ventricle, by the current of blood issuing under pressure through the narrowed mitral orifice during the diastole of the ventricle. Such may be induced by the blood pressure in the auricle and pulmonary veins, independently of the auricular systole ; may be reinforced by contraction of the auricle during the period of diastole, especially at the conclusion of the period; or may be initiated only by the auricular systole just before the contraction of the ventricle.

The mechanism of the presystolic murmur is more complex than that of the systolic, because the factors are more numerous and the conditions more variable. The sounds heard in connection with mitral stenosis can, I consider, be referred to the following types (see Fig. 9) :—

I. Reduplications, or seeming reduplications, of the normal sounds without murmur. I have already (p. 119) called atten-

tion to this as presumptive evidence of mitral narrowing. An apparent reduplication of the *second* sound is the most common. Of thirty-seven cases in which I have made the diagnosis of mitral obstruction, I have found such reduplication in eleven cases. This agrees with the experience of Guttmann (one in three cases) and Hayden (thirty in eighty-one). I have already explained the mechanism of its production. I

Fig. 9.

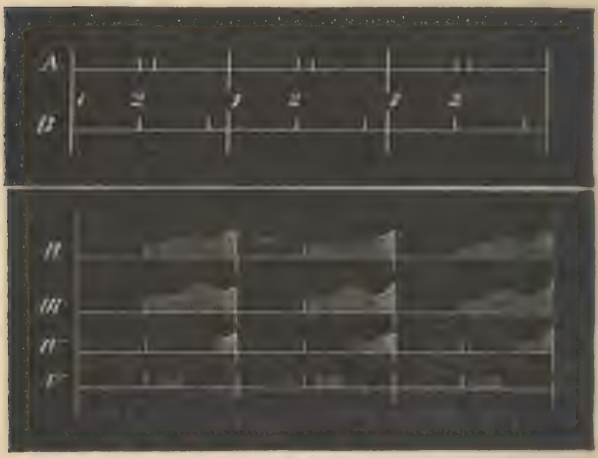

Diagram illustrating varieties of sounds observed in mitral stenosis.

A, reduplication of second sound. *B*, reduplication of first sound. *II.* Prolonged murmur from second to first sound. *III.* The same, with reinforcement during its progress. *IV.* Presystolic murmur, with reinforcement at its close. *V.* Post-diastolic murmur.

believe it to be due to the sudden floating-up of the valve-curtains of the mitral, owing to the increased pressure in the left auricle and pulmonary veins; thus the valve is put on the stretch, and the sound of valve-tension results. Occurring at the earlier part of diastole, this appears like a doubling of the second sound. Occasionally such may be the only ausculta-

tory sign of mitral stenosis, for the presystolic murmur may be absent. We have abundant experience that this murmur may appear and disappear; at the periods of its absence the seeming reduplication may alone be heard. Much more commonly, however, there is a short pause after the second element of the reduplication, and then a murmur of the presystolic character such as we shall presently discuss. In three cases I have noted reduplication of the *first* sound in connection with mitral stenosis. I have discussed the mechanism of this, and have explained it by the view that in these cases the tension of the valve curtains takes place late in diastole—that is, synchronously with the auricular systole. Occurring just before the systole of the ventricle, this gives rise to a seeming reduplication of the *first* sound.

The following types of sound, generated by the conditions of mitral stenosis, are forms of the presystolic (before first sound) murmur.

11. A murmur is heard occupying the whole of the interval between the second sound and the first sound (Fig. 9, *11.*) Usually this murmur receives a distinct emphasis near its termination; it often concludes with a rattle or roll, just as it is terminated by the sudden flap of the ventricular systole. You will observe that the murmur, so far as regards *rhythm*, may be identical with that of aortic regurgitation. In a majority of cases the diagnosis between the two conditions will not be difficult. The area of audibility of the murmur of aortic reflux is usually notably different (as formerly pointed out) to that of mitral narrowing. Nevertheless, it is possible that difficulty may be experienced in the differentiation, for, as I have said, occasionally, though very rarely, the area of audibility of an aortic diastolic murmur is at the apex. In such cases the diagnosis may be peculiarly difficult. My own belief is that the physical signs in the two conditions may be very similar—there may be in each, according to my own observations, a thrill of presystolic rhythm, and in each a seeming reinforcement of the murmur just before the ventricular systole. Indeed, the physical *conditions* in the two cases are very similar. In case of a presystolic mitral murmur such as this, a stream of blood is pouring during the whole period of diastole from the left auricle, and the veins in communication with it, into the left ventricle; vibrations

capable of conveying sonorous impressions are communicated to the ventricle during the whole period, and are intensified at the close when the auricle adds the force of its contraction to the expulsive powers. In case of the aortic diastolic murmur vibrations are in an analogous manner communicated to the left ventricle (in the instance we are now particularly considering to the *apex* of the left ventricle very probably, because the reflux current impinges directly on the upper segment of the mitral valve*) during the whole of diastole, owing to the gushing of the refluent stream through the chink occasioned by the imperfect closure of the aortic cusps, the backward impetus being due to the tension in the aorta and the whole arterial system, *plus* the gravity of the blood column. The physical conditions, therefore, in the two cases are closely analogous—a stream being urged under pressure during the whole period of diastole through narrowed orifice into the left ventricle, and the conduction of the vibrations being to the apex of the ventricle. The difficulty is by no means a merely imaginary one. Dr. Flint has recorded two cases in which murmurs presenting typically presystolic characters had been present, and yet post-mortem examination showed the absence of mitral stenosis and the existence of aortic insufficiency. In regard to these cases, Dr. Flint says, in both "the mitral direct murmur was loud, and had the character of sound which I suppose to be due to vibration of the mitral curtains" (*American Journal of Medical Science*, vol. xliv.) Dr. Charlewood Turner also records a case in which there was a presystolic, alternating with a systolic, bruit at the apex of the heart, together with occasional thrill, and yet the autopsy disclosed no mitral stenosis, but aortic incompetency, the right and *posterior* cusps of the valve being chiefly affected. In another case mentioned by Dr. Turner a presystolic murmur and thrill were perceptible at the apex, as well as a diastolic at the base; here the aortic valves were thickened, though there was no patency (it would seem most probable that there was, nevertheless, some regurgitation during life), and the curtains of the mitral valve were thickened and rough at their margins, though apparently competent. In a third case there were soft diastolic, presystolic, and systolic bruits at the apex,

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* Cf. p. 153.

with a slight diastolic thrill; here, as shown by post-mortem examination, there were aortic disease with incompetency and mitral disease with incompetency, but no stenosis. Dr. Turner does not consider that in any of these cases the murmur, which resembled that of mitral stenosis, was really one of aortic regurgitation, but adduces them to sustain his argument against the auricular causation of the presystolic murmur (St. Thomas's Hospital Reports, 1876). Dr. Hayden mentions a case in which the murmur of aortic reflux simulated that of mitral stenosis ("Diseases of Heart and Aorta," p. 907). It is a significant fact that in almost every case in which a presystolic murmur has been recorded, and the diagnosis of mitral stenosis has not been justified by the autopsy, there has been either a diastolic murmur heard during life in some part of the aortic area, or else the condition of aortic regurgitation has been demonstrated by the post-mortem appearances.

To differentiate between the two conditions, therefore, aid must be called in from other sources than auscultation. The existence of hypertrophy of the left ventricle may be shown by the other signs, and thus the diagnosis will tend to that of aortic regurgitation; but the most important evidence is offered by the sphygmograph and cardiograph (see Part II.). If under such circumstances the diagnosis between the two affections be difficult, the determination of the question of coëxistence of the murmurs of aortic reflux and mitral obstruction may be still more difficult. We shall return to the question when we consider *combined murmurs*.

In the case of the murmur occupying the whole interval between second and first sounds, due to mitral stenosis, I can have no doubt that the early portions of such bruit may be due to the tension in the pulmonary veins and left ventricle, unaided by the systole of the latter. That such could be the case was held by Dr. Wilks. Dr. Galabin came to the like conclusion from cardiographic evidence. The crucial proof of the truth of the proposition is, I consider, furnished by cardiograms taken in cases in which I have found a presystolic murmur to occupy a considerable portion of the diastolic period, and yet the auricular systole is shown to occupy only its usual position just before the systole of the ventricle (see Part II.).

III. A murmur starting after the second sound and ceasing with the first sound is distinctly louder at a certain period in its course. This exaltation of the murmur is indicated by the swelling in the shaded portion of the diagram, which resembles one that was figured by Dr. Gairdner in his early exposition of the presystolic murmur. I have no doubt that this reinforcement is due to the systole of the auricle, which, owing to the obstruction at the mitral aperture, takes place out of the usual rhythm. I shall discuss the evidence which I believe to fully prove that such abnormal action of the auricle can, and does, take place in the section on the graphic signs of mitral stenosis (Part II.).

IV. A murmur occupies a short interval just before the first sound, which abruptly terminates it. This is a common type of presystolic murmurs, and is due chiefly or wholly to the communicated impetus of the auricular systole (see Part II. p. 272).

V. A murmur is affixed to the second sound, but ceases before the commencement of the first sound. Such murmur has been described by some observers as diastolic, by others as post-diastolic. The strictly diastolic form of murmur generated at the mitral orifice is very rare. Dr. Hayden states that he has not met with an example, but mentions a case under the care of Dr. Stokes in which it existed together with a systolic murmur. I have recorded a similar case, with what I consider to be the explanation of the phenomenon in that instance. Dr. Fagge has mentioned cases in which a post-diastolic murmur of mitral causation ceased before the commencement of the first sound, and it is quite conceivable that in such the tension in the left auricle was sufficient to produce a murmur as soon as the relaxation of the ventricle allowed the flow of blood to occur into it; but the muscular feebleness of the auricle was such that the murmur was not only not prolonged, but at the later period not even produced. In some rare cases the two last-mentioned murmurs have been combined: there is "a murmur of double rhythm, or broken into two fragments, one of which adheres as a prefix to the first sound, and represents the ordinary presystolic murmur, whilst the other succeeds the second sound, being appended to it as a *suffix*: these two fragments being separated by a brief period of silence" (Dr. Hayden).

We may now briefly consider COMBINED MURMURS. We have already discussed double murmurs—that is to say, murmurs generated at different periods of the heart's rhythm at one orifice: by "combined" murmurs I mean those which take their origin from more than one of the orifices. The following may be taken as probably representing the relative frequency of the combinations:—

(1.) Mitral regurgitation and aortic obstruction.

(2.) Mitral regurgitation and aortic regurgitation, or mitral regurgitation and aortic obstruction and regurgitation.

(3.) Mitral obstruction and aortic regurgitation, or mitral obstruction and regurgitation and aortic obstruction and regurgitation.

(4.) Mitral obstruction and regurgitation with tricuspid obstruction.

(5.) Mitral obstruction and regurgitation with tricuspid obstruction and regurgitation.

(6.) Aortic obstruction and pulmonic obstruction.*

None of these combinations require particular comment from the point of view of diagnosis, except the third. You will distinguish them by the rules already laid down for the diagnosis of the individual affections. In all cases notice: 1. The positions of maximum intensity of any murmurs heard over the cardiac area. 2. Any differences of pitch and character. 3. The directions in which the sound is conveyed.

There may be considerable difficulty in determining

* The combination of mitral regurgitation and tricuspid regurgitation, or mitral obstruction and tricuspid regurgitation, is a very common one, but very frequently the tricuspid lesion is not betrayed by *murmur*.

the co-existence of aortic insufficiency and mitral narrowing. A prolonged diastolic murmur may drown the presystolic. In carrying the stethoscope, however, down the left border of the sternum you may probably arrive at a spot where the diastolic murmur ceases to be audible, and then as you approach the apex, a presystolic of different pitch and character may become manifest. The presence of a presystolic thrill at the apex may aid the diagnosis.

Even though the ordinary means of diagnosis be used with all care, the question whether or no a mitral direct co-exists with an aortic regurgitant murmur may be difficult to determine. Witness the following cases under my care :—1. A man of fifty-one, in whom was diagnosed, from the physical signs, obstruction and regurgitation both at mitral and aortic orifice. There was no doubt concerning the signs of aortic obstruction and regurgitation; in addition, there was "a rolling presystolic murmur two inches below and an inch outside the nipple, this murmur terminating in a blowing systolic bruit conducted to axilla." The sphygmographic signs were typically those of aortic reflux. 2. Alice B., aortic diastolic murmur with presystolic murmur presenting usual characters. Sphygmographic signs of free aortic reflux. Cardiograph shows no sign of mitral stenosis. 3. H. M., low-pitched murmur with first sound at aortic cartilage; second sound murmur left of sternum; rolling *typical* presystolic murmur at apex. Cardiograph shows much hypertrophy of left ventricle, but no sign whatever of mitral stenosis. 4. Walter F., aged twenty, murmur at first heard in mitral area, commencing very shortly after second sound, and after augmenting in intensity, ceasing abruptly with first sound; in fact, closely resembling murmur of mitral stenosis. Coarse thrill also felt at apex. Afterwards, murmur heard to be diastolic down left border of sternum, but still typically presystolic at apex. Cardiographic and sphygmographic evidence indicated free aortic regurgitation, but no sign of mitral stenosis.

I cannot think that in any of these cases, though there was a murmur which, in the absence of aortic regurgitation, I should have considered pathognomonic of mitral stenosis.

there was any other valvular lesion than aortic insufficiency. My belief is, as I have before indicated, that the murmur of aortic reflux may so closely simulate that of mitral obstruction as to be absolutely indistinguishable from it. It follows that if you meet with a case in which, from the co-existence of the signs of the two conditions, you are inclined to make a diagnosis of the two lesions, it is right that you should hesitate and consider all the available evidence. Several of the cases which have been adduced as telling against the auricular causation of the presystolic murmur have been where the signs of aortic reflux have been undoubted. Now the combination of mitral stenosis and aortic regurgitation, as shown by post-mortem records, is a rare one; that of mitral regurgitation with aortic regurgitation being much more common. In my belief, the presystolic murmur heard in aortic regurgitation does not invalidate the auricular hypothesis, but, as I have said, the physical conditions are so analogous that the sonorous results may be identical. The quasi-presystolic murmur of aortic regurgitation is the concluding portion of a diastolic murmur heard at the apex; if the tension is great in the aorta, at the end of diastole, the murmur is generated with equivalent force to that exercised by the auricle in the mitral lesion; moreover, the backward flow is capable of generating thrill, which, being most evident at the later periods of diastole, seems to be presystolic.

I have never met with a case of co-existent mitral and tricuspid stenosis, but Dr. Hayden, of Dublin, has recorded three such examples. The presence of tricuspid stenosis is to be suspected whenever a presystolic murmur is heard close to the left edge of the sternum. Dr. Hayden "would regard the existence of two centres of presystolic murmur, with or without fremitus—viz., at the apex and somewhat to the right of that situation, in conjunction with marked systemic venous engorgement—as evidence of the double lesion of mitral and tricuspid stenosis."* I

* "The Diseases of the Heart and of the Aorta," by Thomas Hayden, &c. &c. (Dublin: Fannin & Co., 1875), p. 238.

had one case which seemed to present this combination, but the murmur simulating that of tricuspid stenosis was found to be due to a rough patch of pericardium over the right auricle. The probabilities of tricuspid stenosis were great, for there was very marked presystolic pulsation in the veins of the neck. There was tricuspid regurgitation.

The diagnosis of complex pathological conditions of the heart sometimes presents very considerable difficulties, and requires great care and repeated examinations. I would venture to give you one or two rules to observe when you meet with a difficult case.

In the first place, do not be content to write in your notes, "rhythm of the heart irregular and tumultuous," but let there be order in your record of such irregularity, and system in your treatment of the seeming chaos.

Record all the signs which you have observed previously to those derived from auscultation.

Describe the sounds, normal and abnormal, heard over the situations of each of the orifices.

Note, first, the characters of first sound and second sound at the aortic cartilage. Reduce these to diagrammatic form (see p. 197 *et seq.*), indicating murmurs where present.

Repeat the process at the pulmonary, tricuspid and mitral areas successively.

Compare the observations and diagrams only after they have been completed, and then fill in the lines of conduction of normal and abnormal sounds (see p. 83).

Do not unduly hasten to form your conclusions, but obtain all the evidence possible before you give your verdict.

PART II.

THE SPHYGMOGRAPH

AND CARDIOGRAPH.

PART II.

THE USE OF THE SPHYGMOGRAPH AND CARDIOGRAPH IN THE DIAGNOSIS OF DISEASES OF THE HEART.

I.

Invention of the Sphygmograph—Illustrations of its usefulness—Varieties of instruments—Mahomed's Sphygmograph—Pond's Sphygmograph—Galabin's Cardiograph.

THE sphygmograph is an instrument for enabling the movements of an artery which constitute the pulse to be automatically recorded; the term cardiograph is used for an appliance which writes the motions of the heart itself. The idea of making the pulse register its own movements dates from long-past ages: it is said to have occurred to Galileo. Anything like perfection in the mechanism adopted has, however, only been obtained in recent years, when Marey devoted himself to the investigation of the phenomena of movement in the vital functions. His sphygmograph is the model on which other instruments have been constructed. The mechanical difficulties of record being for the most part overcome, there remain difficulties of interpretation—the pulse writes its own hieroglyphics, but these have to be expounded. The interpreter obtains the key by patient observation and by experiment. One advantage of the evidence obtained by such mechanical means is that, whilst the impressions we have gained by the

methods of physical investigation hitherto considered —whether those derived from our sight, touch, or hearing—may fade into the haze of half-forgotten memories, the story which the pulse writes of itself is permanent. "Litera scripta manet."* You may ponder over the record, solve its intricacies at your leisure, and compare the evidence obtained at one time with that procured at another. But the question is asked, Are these *practical* instruments of diagnosis? If by diagnosis we mean not merely the detection of definite diseases, but the estimation of the degree of deviation from standard health, undoubtedly they are. Is the value of the indications which they give commensurate with the trouble which must be taken to employ them? Under present circumstances, certainly. Thanks to the ingenuity of physicians who are also mechanicians, the difficulties are so far overcome that the instruments may be used with very little expenditure of time: they are of daily employment, and every day gives evidence of their value and importance. They should be used, however, after the other means of diagnosis, not before. As Dr. Mahomed has said, they open up no "royal road to diagnosis," but they may check, confirm, or extend the diagnosis already attained, and often throw a new light upon a difficult position. I may illustrate the value of the sphygmograph by two examples. I was called hurriedly to a patient whom I found in the extremities of cardiac dyspnœa. She was unable to lie down; there was extensive œdema, and a loud systolic murmur was heard at the apex of the heart, conducted towards the axilla and audible at the

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* "Segnius irritant animos demissa per aures
Quam quæ sunt oculis subjecta fidelibus."—HORACE, *Satires*.

back. I had no doubt that the condition was that of mitral regurgitation. There was, however, in addition, extreme anæmia, with hæmic murmurs at the base of the heart and over the vessels of the neck, and it was probable that this had been brought about by excessive hæmorrhage caused by uterine fibroid. The question which now presented itself was—Is this cardiac dyspnœa with mitral regurgitation due to organic disease of the valve, or is it the result of weakness of the muscle of the ventricle? Against the first view was the fact that there had been no rheumatism, acute nor subacute. On looking at the pulse-tracing, which I had at once obtained, I was struck with the fact that it so nearly approached the normal; I concluded, therefore, that the imperfection of the mitral orifice was *not* the result of valvular disease. This diagnosis proved correct, for after treatment the whole of the cardiac signs and symptoms passed away, and no trace of the murmur at the apex remained. Another instance. I was called in consultation with a medical friend to the case of a gentleman, past middle-age, who in appearance was hale and well-nourished, and who, it was well known, had led a careful, abstemious life. The signs were those of a limited pneumonia of the base of the right lung. The general conditions did not suggest a bad prognosis, the only reason in favour of which was the asserted debility of the patient on attempted movement, even while in bed; an expression which seemed not to be corroborated by the appearances. To the finger the pulse showed a fair regularity. The sphygmographic trace, however, gave a different lesson; though there was fair regularity in *time*, there were such variations in volume and in general characters that I feared the heart-fibres were degenerated. The prognosis

founded on this observation was gloomy, and the probabilities which were then sketched out were exactly confirmed by the facts. The signs of pneumonia passed away, and it appeared that there might be a good recovery; then ensued a sudden syncope and a situation of grave peril. From this there was a rally and a renewed hope of recovery; syncope, however, in spite of the most watchful care and abundant nourishment, recurred, and the patient died. These two instances are, I think, sufficient to show the value of the sphygmograph as an instrument of diagnosis: in the one case I consider that it determined the difficult question whether there was organic disease of the mitral valve or passive yielding of the ventricular walls; in the other it demonstrated an unsuspected condition of degeneration of the muscular fibres of the heart. I propose now to examine more systematically its application and its uses in the diagnosis of cardiac diseases.

Varieties of Instruments.—I have said that these are chiefly founded upon the model of Marey. The modification of Marey's sphygmograph usually employed in this country is that designed by Dr. Mahomed.

Mahomed's sphygmograph (Fig. 10) consists essentially of a framework containing an arrangement of levers adaptable to the pulsating vessel. Contact with the artery, the radial being usually chosen for convenience, s made by a steel mainspring, the movements of which, consentaneous with those of the artery on which it is made to rest, are communicated to a lever of the third order, so that the slight motion may be much amplified (about ninety times). The writing lever consists of a fine slip of very light wood, and is so guarded as to allow vertical without lateral movement. The motions

of the pulse are thus converted into up-and-down strokes of the free extremity of the lever; these are made to impinge upon a slide, which, by means of clockwork, is caused to travel at a known rate—about

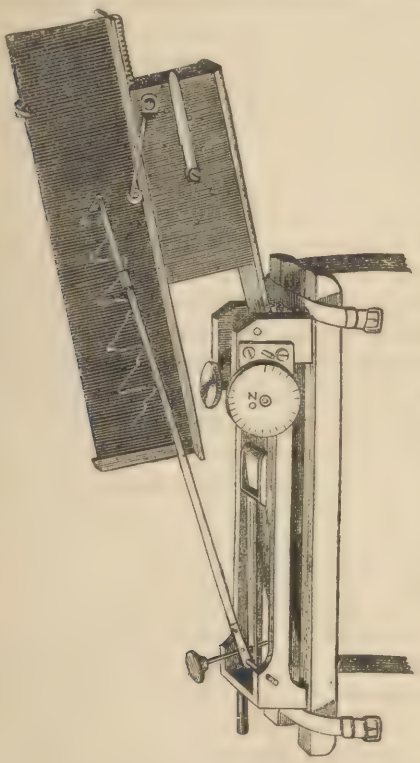


Fig. 10. Marey's Sphygmograph, improved by Mahomed.

four inches in ten seconds. Thus the movements of the free extremity of the lever, representing the amplified movements of the pulse, are described upon the travelling slide in a series of figures. For a

minute description of the instrument I must refer you to Dr. Mahomed's paper (*Medical Times and Gazette*, January 20, 1872, p. 63).

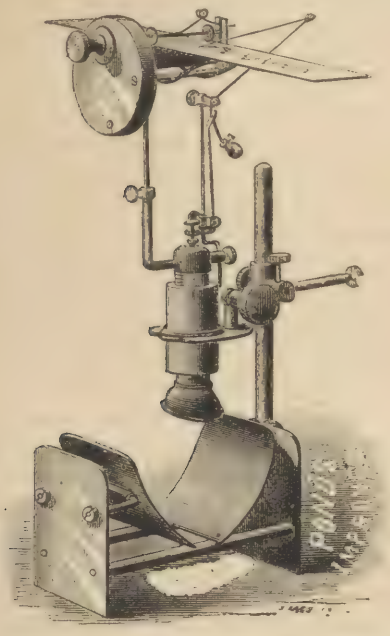
An essential point is to be able to apply the main-spring to the artery with definite degrees of pressure. This object Dr. Mahomed has attained with great success. By a very ingenious arrangement the pressure can be increased by turning a thumb-screw, and this pressure is registered in ounces (1 to 18 troy) upon a dial-plate.

Pond's Sphygmograph (Fig. 11), which is also adapted for use as a cardiograph, has the great merit that it is very readily applied, and its application consumes so little time that it may be used in daily practice. I believe that every practitioner, however busy, would be able to employ it, and would find its indications of the greatest possible service. In this instrument the slide of paper or of mica on which the tracing is taken, instead of being vertical as in the other instruments, is horizontal, so that the operator looks down upon it. The throbbings of the pulse are communicated to an india-rubber diaphragm, or, by a late improvement of the instrument, to a vertical lever terminating below in a small metal button, and kept in constant contact with the artery by a spiral spring. A very ingenious, yet simple, arrangement of levers causes amplification of the movements which are recorded on the slide, which, as in the other instruments, travels by clockwork (about six inches in fifteen seconds). The upright stem of the instrument is provided with a pressure-gauge graduated from one to sixteen ounces, so that various degrees of compression can be exerted upon the artery

The sphygmographs of Marey and Mahomed have

been used for taking tracings directly over the heart's impulse, but, in many cases with these instruments, the task is impossible—first, because with a strongly-acting heart the range of motion, amplified as it is by the sphygmograph, is too great to be recorded on

Fig. 11.



Pond's Sphygmograph and Cardiograph.

the travelling slide; and secondly, because the mechanical arrangement adapted to the limited area of a pulsating artery is ill fitted for fixing over the chest-wall at the site of the heart's impulse. Marey first overcame the difficulties, and laid the foundation for

the study of the movements of the heart by the graphic method, by constructing a cardiograph in which the cardiac motion of the chest-wall was transmitted to a drum or tympanum, communicating by a flexible india-rubber tube with a second tympanum, the two tympana, and the tube which connected them, forming an air-tight cavity. Thus the motion imparted was transmitted by the oscillations of the column of contained air; it was amplified by a lever attached to the second tympanum, and recorded on a revolving cylinder termed a polygraph. Many observations of the highest importance were made by means of this instrument, but it is now superseded by a modification of the sphygmograph adapted to the chest-wall.

GALABIN'S CARDIOGRAPH is that generally adopted in this country. Its general construction is that of a magnified sphygmograph, with adjustments for its adaptation to the thorax. Instead of the rigid parallel bars of the sphygmograph, the base is constituted by moveable wooden blocks, which can be separated to a width of nearly five inches; by means of these the instrument is made to rest upon the chest. Between them, by two transverse steel rods, the brass frame of the cardiograph is supported, and is attached, by joints which allow of both vertical and horizontal adjustment, to four vertical rods. It is possible, therefore, for the instrument to be applied to a chest of any size or shape. The brass frame differs from that of the sphygmograph in that the bar attached to the main-spring has an arrangement for its elongation and variation of position, so that the vertical height of the curve described by the recording style can be varied from 10 to about 100

times the actual movement of the chest-wall. The mechanism for an exact determination of the pressure exerted by the main-spring is unnecessary in the cardiograph, but there is an adjustment for reducing this pressure to nearly zero, so that very slight impulses—even the back-stroke in veins—may be recorded. (For Dr. Galabin's description *vide* "Med. Chir. Trans.," vol. lviii. p. 353.)

II.

Method of obtaining a sphygmographic tracing with Mahomed's sphygmograph — Importance of recording the pressure employed—Tracings with Pond's sphygmograph—Cardiographic tracings with Galabin's cardiograph With Pond's instrument—Varnishing tracings for preservation—To multiply copies of tracings.

How to obtain a tracing—(1) with *Mahomed's Sphygmograph.*—The arm of a patient from whose radial artery a tracing is to be taken must be placed upon a splint or rest, the palm being upwards. The stand of the instrument is then applied over the arm in the longitudinal direction, so that the ivory pad of the mainspring is just over the artery as the latter lies on the inner side of the styloid process of the radius, and is crossing the anterior ligament of the wrist-joint. In this position the instrument is firmly maintained by strong bands passing over each extremity of the framework. The hand is to be bent slightly backwards, and may be made to gently grasp a band stretched across the end of the splint or pad. If the wrist be strongly extended backwards, the parts are too tightly stretched and the artery rendered tense; if the hand be firmly closed, the tendons of the muscles are tightened and made to interfere with the perfect application of the instrument, or, perhaps, to produce vibrations in the tracing. The instrument being applied with these precautions, the artery will now lie between the ivory rods, and the pressure of the spring upon it must now be varied (by the operator

turning the graduated thumb-screw) until the maximum range of movement of the recording lever is attained. The "slip" upon which the tracing is to be taken is now placed on the travelling slide. This slip is generally of white paper, which should be well glazed on both sides, so that friction is reduced to a minimum. The style of the recording lever may be supplied with ink, so that it writes upon the white paper as the clockwork arrangement causes it to travel along; this method, however, is often tiresome, the pen refusing to mark or the ink being jerked into unsuitable places. The best plan appears to be to allow the point of the style to make its mark upon the paper-slip, which has been previously smoked. In order to smoke the slip it is only necessary to hold for a brief period the paper over a small piece of burning camphor, a kindled pledget of cotton soaked in olive oil, or a smoking paraffin lamp: by either of these methods a uniform layer of black is obtained over the surface of the paper, and, of course, the end of the writing-lever makes, by displacing the smoke, a series of white markings as it moves over the surface. The blackened slip being introduced, and the clock movement wound up, a touch of the button is made to liberate the spring, and the slide commences to travel. It may be stopped at any time, and then the pressure varied. The most perfect tracing is that in which, the spring following most perfectly the expansion of the artery, the summit is most sharply defined (*i.e.* free from rounding) and the various notches best marked. Dr. Mahomed says: "The use of incorrect pressure is perhaps the most common source of error in sphygmography, and the greatest care is necessary in deciding upon the amount of pressure to be

employed. This, when ascertained, should be carefully recorded, as it is one of the most important characters of the pulse. Notice should also be taken of the amount of pressure required to totally extinguish the pulse, so that the lever remains perfectly motionless; for by this means an estimate can be formed of the force with which the heart is contracting."* A number of tracings can of course be taken at one sitting: they may be at once examined with care lest manipulation rub off the coating of smoke. The name of the patient, &c., the pressure employed, with a brief note of physical signs, should then be inscribed, a needle or finely pointed steel pen being used for this purpose, and each slip be varnished for preservation.

(2.) Tracings with *Pond's Sphygmograph* are thus obtained. It is not necessary to use a pad or splint, but a metallic wrist-rest is supplied with the instrument: this is ingeniously constructed, so that it adapts itself to an arm of any size with no discomfort to the patient. Tracings may, however, be taken even without the wrist-rest. An upright bar is inserted into this support, to which is clamped a transverse arm, which clips the body of the sphygmograph; this arm is provided with a screw adaptation to all necessary positions. The patient being in the sitting or recumbent posture, and quite at ease, the hand rests with palm uppermost; then—the slip of smoked paper or mica having been introduced by momentarily setting free the watch movement, allowing the slip to travel for about a quarter of an inch, and then stopping again—the operator, holding the sphygmograph by the cylinder.

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* *Medical Times and Gazette*, Jan. 20, 1872, p. 64.

applies the india-rubber base over the pulsating artery. Due contact between the upright bar immediately in relation with the india-rubber diaphragm, and the system of writing levers must now be made by sufficiently turning the small screw at the summit of the cylinder. The writing-needle will now be observed to move over the blackened slide. By slightly varying the position of the india-rubber base over the artery, and by increasing or decreasing the firmness of application of the instrument (this pressure being determined by noting the pressure-gauge), the point of maximum movement of the needle will be observed. If the attachment to the wrist-rest be employed, the instrument should be fixed in the position at which this maximum of movement is attained; but with a little practice the operator will be able to retain the sphygmograph with uniform pressure upon the artery, and to dispense with the clamp which fixes to the wrist-rest. Then the watch movement is liberated by a touch of the finger, and the slide travels. Several tracings should be taken at varying pressures. It is very easy to obtain tracings with this instrument from the carotids, the temporal arteries, the femorals, &c., as well as the radials.

I consider it best, when using Pond's instrument, to take the first slip with gradually increasing pressures every three or four pulses. Commence with the lowest possible pressure that will develop the tracing, then stop the travelling of the slide, and start it again, using slightly increased pressure; observe whether increasing pressures increase the altitude and development. Note the pressure which gives the maximum, and the effect of higher pressures on the elements of the trace. Often one slip suffices, but it may be necessary to take two or three. Thus you are informed at what pressure you have the best tracing, and can proceed to take others at this, which gives you the most typical results.

(3.) Cardiographic tracings may be thus obtained with *Galabin's Cardiograph*. The patient being stripped to the waist, the position of the apex-beat should be determined and marked upon the chest-wall by means of a pencil. The pad of the lever is then to be applied over the point of pulsation, and the instrument adapted by separating to greater or less degree the wooden blocks constituting the base, as well as by altering the position of the instrument in relation to the vertical rods until it rests suitably upon the chest. It may be fixed by two partially elastic straps passed round the body and fastened by buckles. In many instances it suffices to hold the instrument against the chest with the hand, the straps, &c., being dispensed with. The movements of the recording style being observed to be in suitable position upon the smoked paper, the clockwork is started and the tracing taken. In some cases the movements of respiration do not greatly disturb the position of the style, and the series of tracings is well comprised within the slide. Often, however, the movement of breathing causes too great excursion of the style, and the patient must be asked to hold the breath—the clockwork being started at the moment of arrest at the end of expiration. The position of the patient should be as much as possible one of ease: if the apex-beat be fairly defined, it should be that of as complete recumbency as is consistent with comfort; if the apex-beat be ill-defined, a better tracing may be taken if the patient be in a sitting position, leaning somewhat forwards.

(4.) Cardiac tracings with Pond's instrument are often obtained with great ease. The preliminary arrangements having been made as when it is used

as a sphygmograph, the instrument is simply held against the chest-wall at the point of pulsation, and the clockwork started. To obtain a satisfactory tracing it is usually necessary for the patient to be recumbent; but it is possible to obtain one in the sitting and even standing posture. The sphygmograph is held by the cylinder or by the barrel of the watch movement; the difficulty is, that the needle shall not by the force of gravity fly off the smoked surface; sometimes this difficulty may be obviated by removing the little weight which serves as a counterpoise to the needle, sometimes also by bending the needle itself. It is very important that there should be facility for taking tracings not only in the sitting position, but even when the patient leans well forward.

To preserve sphygmographic and cardiographic tracings they must be *varnished*. Before this is done, upon each tracing should be inscribed the name and age of the patient, and a brief note of the physical signs or the diagnosis arrived at by the hitherto employed means. This is easily done by writing on the smoked surface with the point of a pin or a needle fixed into a convenient handle. The tracing should then be carefully dipped in a rapidly drying varnish. The varnish may be composed of gum benzoin and methylated spirit (one ounce of the former to six ounces of the latter). The following is Pond's receipt:— Alcohol, one pint; gum sandrac, three ounces; castor-oil, half-ounce; mix.

To multiply copies of tracings the original should be taken on a slip of mica and well varnished. When quite dry it should be placed in a pressure-frame, such as used by photographers, with a slip of photo-sensitive paper behind it, so that the latter becomes printed off

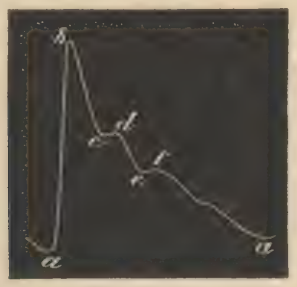
as a positive impression from a negative photograph. A child's transparent slate provided with a clear glass forms a very efficient pressure-frame. The solution with which the paper is rendered sensitive is thus prepared:—(1.) Ammonio-citrate of iron, $1\frac{7}{8}$ ounce; water, 8 ounces. Make a solution. (2.) Red prussiate of potash, $1\frac{1}{4}$ ounce; water, 8 ounces. Dissolve. Mix the two solutions and keep in the dark or in a yellow bottle. This solution being poured into a saucer, strips of white unglazed paper are to be wetted with it on one side only, and then allowed to dry in the dark until wanted. The sensitive side of the paper being pressed against the negative mica-slip, so that the tracing reads correctly, it is exposed to sunlight or daylight for a term varying from twenty minutes to two hours according to the intensity of the light. When removed from the pressure-frame, the paper must be washed in clear water for a minute or two, and the tracing will appear in blue lines upon a white ground. If properly managed, the copy is permanent. It is obvious that copies of many slides may be taken in one frame at the same time, and by consecutive operations almost any number may be procured from one mica.

III.

Interpretation of the normal pulse-trace—The first event, or percussion wave—The second event, or tidal wave—The third event, or dicrotic wave—The diastolic portion—Interpretation of the normal heart-trace—The systolic portion—The diastolic portion—Indications afforded by the sphygmograph—Frequency of pulse—Irregularity in time—Intermittency—Irregularity in volume, rhythmical and arhythmical—Relation of pulse to respiration—Respiratory line—Undulation of base-line in cerebral diseases.

Interpretation of the normal pulse-trace.—The tracing of a single pulsation in a state of health consists of (1) a line which is vertical or nearly so (Fig. 12, *a b*).

Fig. 12.



The normal pulse-trace magnified: *a b*, percussion upstroke; *a b c*, percussion wave; *c d e*, tidal wave; *d e f*, dicrotic wave; at *e*, aortic notch; *f a*, diastolic period.

The contraction of the left ventricle forcing the contained volume of blood into the aorta, suddenly dis-

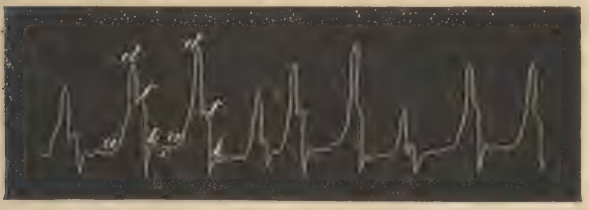
tends the systemic arteries; the lever, therefore, in contact with the artery, receives a sudden impulse which jerks it upwards; the vertical line is the result of the movement of the vessel thus produced, amplified by the mechanism of levers and somewhat by acquired velocity. This, then, is the *first event*, an almost instantaneous up-stroke, often termed the percussion-stroke. Having attained its maximum height, (2) the line now begins to descend at an acute angle to the up-stroke, the sudden force of elevation having been expended, but again rises at *c d*, so that it forms a curve. This is due to the fact that the lever, which falls by its own weight after the expenditure of its abruptly-communicated velocity, is again caught and lifted by the tide of the onward current of blood. This is the *second event*, and the wave thus produced is termed the *tidal wave*. The line inscribed by the lever now descends, and again rises, forming a second curve (*e f*). This is the *third event*, the *dicrotic wave*. The curve produced is an important one. It is due to a rebound of the blood from the *point d'appui* constituted by the cardiac end of the aorta with its closed semilunar valves. Marking as it does the closing of the aortic valves, the notch thus produced is termed the *aortic notch*. So far, therefore, the trace has indicated only the systolic portion of the cycle; the brief remainder comprises the diastolic portion. This consists of the wave springing from the aortic notch and a gradually sloping line, ending at the base-line of the tracing. Its vertical height is about one-third of the percussion up-stroke. In delicate tracings a very slight elevation is sometimes seen just as the down-stroke ends and the up-stroke of the succeeding pulse commences; this marks the systole of the auricle.

Interpretation of the normal heart-trace (Figs. 13 and 14).—As with the sphygmogram, the most prominent feature of the cardiogram is a sudden elevation denoting the contraction of the ventricle. Following

FIG. 13.

The normal cardiac trace (after Galabin). *A*, Typical apex tracing. *a*, auricular systole; *a d*, ascent due to sudden hardening of ventricles; *d e f*, continued systole; *k*, rebound after emptying of ventricles; *l*, ascent from gradually increasing influx of blood into ventricles during diastole. *B*, Tracing from a healthy man; tension low, and heart acting vigorously. *g*, notch indicating closure of semilunar valves.

FIG. 14.



Normal cardiac trace (after Pond). The references are the same as in Fig. 13.

the chief elevation, on the broad summit of the trace, however, are one or two waves which have been said to denote oscillations of the auriculo-

ventricular valves during systole. It seems to me more probable that they are due to the muscular movements of the ventricle. At the end of the elevated portion of the trace is a more rounded swelling, which marks the termination of the ventricular systole. The line then descends with a slight slope to the base-line. Now commences the diastolic portion of the tracing. In this is seen first an eminence, which indicates a shock lifting the apex after the relaxation of the ventricles; next, a slightly ascending line, broken by some slight undulations, marking the time during which the blood-pressure in the ventricle is increasing; and lastly, a small eminence immediately before the succeeding up-stroke, due to the contraction of the auricle.

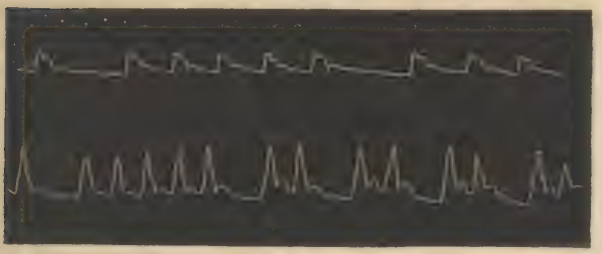
It will be seen that the evidence obtained by the cardiograph is complementary of that afforded by the sphygmograph. By the latter we obtain a more complete record of the systole of the ventricle, and its effect on the peripheral arteries, whilst by the former we gauge more completely the conditions of blood-pressure in the ventricle during diastole.

INDICATIONS AFFORDED BY THE SPHYGMOGRAPH.—The sphygmograph records the *frequency* of the pulse. Care should be taken as regards each instrument to estimate the time which the slide takes to travel. This may be done by a stop-watch, and the mean of several observations should be taken. Occasionally the process should be repeated with individual instruments to ascertain whether in course of time the clockwork alters speed. The instrument is usually so constructed that six inches travel in fifteen seconds. To count the pulse, therefore, measure off six inches of the tracing, and multiply by four the number of

pulsations observed. This of course gives the rate of the pulse per minute.

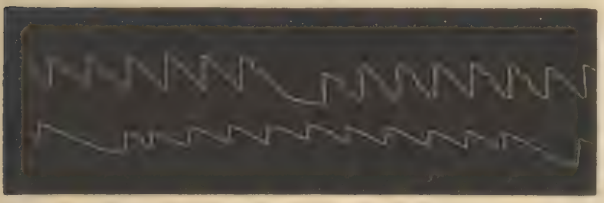
The sphygmograph also records *irregularities in time.* It registers *intermittency* in the pulse (see Figs. 15 and 16). It is obvious that in these cases the heart rests

FIG. 15.



Irregularities in time. Tracings from right radials in cases in which irregularity disappeared after treatment. (In the upper tracing tension is moderate; in lower tension is low.)

FIG. 16.



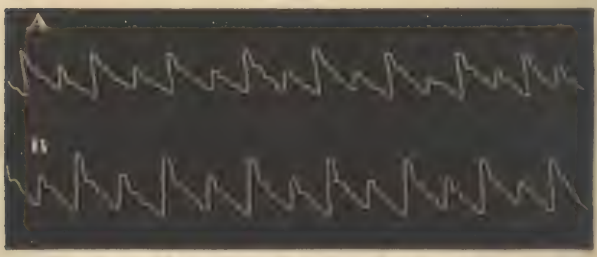
Intermittency in pulses of high tension.

during the period of a single pulsation, and that this intermittency is repeated at intervals. Such irregularities are not uncommon in old persons, or in the young when convalescent from acute diseases. They are usually of no serious prognostic import. Sometimes

the condition is habitual, and uninfluenced by treatment; in others it is remedied with ease. A change of dietary sometimes alone suffices; abstinence from tea, coffee, or other exciting beverages, may soon restore the heart's regularity. In the case of one of my patients the irregularity entirely ceased after omitting potatoes as an article of food; no doubt the error of excess in this particular had been committed previously. The indication, however, may be very important; in one of my patients the advent of intermittency occurred in the early stages of the development of cancer of the liver, when the signs and symptoms were very obscure.

A glance at the sphygmographic tracing shows also if there be *irregularities in volume*. These may be *rhythmical* or *arhythmical*. The most striking example of rhythmical irregularity of volume (Fig. 17) was in a case in which there was no cardiac disease, but hemi-

FIG. 17.

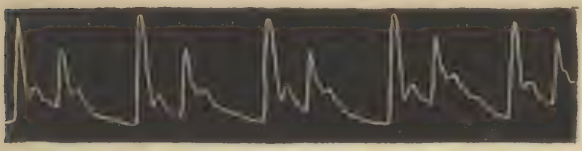


Rhythmical irregularity in volume in a case of cerebral disease attended with Cheyne-Stokes dyspnœa. Traces of right radial artery (5 oz. pressure). *A*, taken during period of excitement of respiration; *B*, during arrest of respiration.

plegia, aphasia and the phenomena of Cheyne-Stokes respiration (see p. 35). The autopsy showed that there was much cerebral disorganization, the remote effect of

embolism of some of the cerebral arteries. In my opinion, there was in this case a direct disturbance both of the respiratory and the cardiac centres. The pulse-tracing shows that each systole of greater is succeeded by another of lesser amplitude (Fig. 17).* Another example of double systole (the *pulsus bigeminus*) is seen in a tracing by Dr. Pond (Fig. 18).

FIG. 18.



Pulsus bigeminus.

Irregularities in volume in which there is no observance of rhythm as indicated by the sphygmograph are of high importance both in diagnosis and prognosis. Here we observe the elevations in succeeding pulsations to vary very considerably, and in an irregular manner; so in the trace the height of the percussion stroke in one instance may be four times greater than in others. *Arhythmical irregularity in volume* may be demonstrated by the sphygmograph when it is not detected by the finger. The tracing (Fig. 19) was taken from a case in which the finger failed to detect any notable irregularity. This was no doubt due to the fact that the pulsations were fairly regular in point of *time*. The sphygmographic evidence alone led to the diagnosis of cardiac degeneration.

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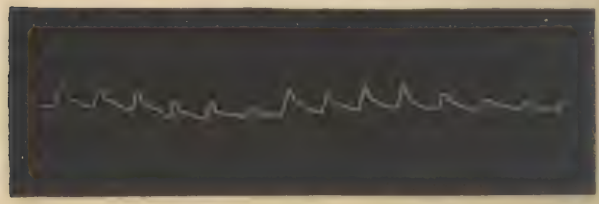
* This has been termed by Traube the "pulsus alternans."

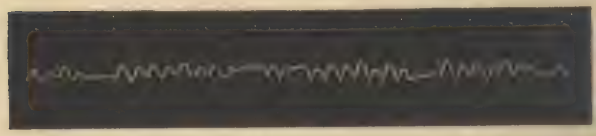
Irregularities in both time and volume are shown in the acute fevers when the muscle of the heart has

FIG. 19.



Arhythmical irregularity in volume. Tracing from right radial artery in a case of pneumonia, with probable cardiac degeneration.

FIG. 20.



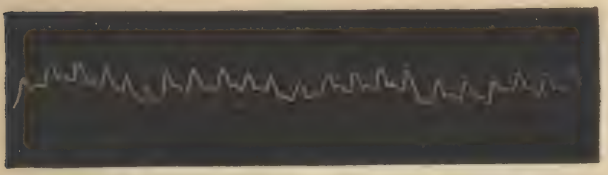
Irregularity in time and volume (myocarditis in course of typhoid fever).

been involved in disease (Fig. 20), as well as in the later stages of valvular diseases.

Inspection of the sphygmographic trace in series also indicates *the relation of pulse to respiration.* The base-line of a series—that is to say, a line drawn so as to join the lowest points of the percussion up-strokes—should be straight. A line so drawn is termed the *respiratory line.* If the arterial tension vary, this line will be not straight but a series of curves. In health, if a deep inspiration be made during the time that the sphygmogram is taken the line curves downwards, because, owing to the suction-action of the thorax, general arterial tension is reduced; on the

other hand, if a deep expiration be made, an elevation takes place in the respiratory line. In cases of disease in which the rhythm of respiration is disturbed, this curving or undulation of the base-line is observed, and as each curve represents a respiration, the ratio of pulse to breathing is easily ascertained by counting

Fig. 21.



Marked undulation of base-line (from a case of tubercular meningitis).

the number of traces in each curve.* In other cases there may be seen irregularities in the curves not corresponding to the obvious rhythm of respiration: thus one pulse-trace in every two, three, or five, may descend to the base-line; this is especially seen in cases where there are *cerebral* complications. (See Fig. 21.)

* Such a trace is seen in Fig. 25.

IV.

Interpretation of the abnormal pulse-trace—Causes of augmented and of lessened percussion wave—Oblique percussion—Causes of increase and of decrease of tidal wave—Of dicrotic wave—Estimation of arterial tension—Pulses of high and of low tension—Supernumerary vibrations.

INTERPRETATION OF THE ABNORMAL PULSE TRACE. The elements which we have considered of the pulse-trace are all capable of modification under conditions of disease. The percussion-wave is increased by *sudden and forcible contraction of the left ventricle, and by a large amount of blood distributed by the systole.*

FIG. 22.

Trace showing oblique ascent and descent (tidal wave only), taken from left radial artery in a case of aneurism of the left subclavian (Dr. B. Foster).

It is lessened by feeble or gradual contraction of the ventricle, and by a small amount of blood distributed by the systole. When the up-stroke deviates from the vertical line, there is toiling on the part of the ventricle, or disease in the coats of the vessel through which the arterial current flows. In some cases in which the sphygmograph can be applied directly over the sac of

an *aneurism* the tracing shows only gradually sloping lines of ascent and descent; it is thus reduced to its simplest form, resembling that recorded by the mere pulsatile injection of fluid into an elastic tube. There is here only one wave—the tidal; it is exemplified in the tracing (Fig. 22) taken from the left radial in a case of aneurism of the left subclavian artery by Dr. Balthazar Foster.* The aneurismal sac here acts the part of a mere elastic bag. In other instances the modifications are less notable, but a sloping line of ascent is characteristic (see Fig. 23).

FIG. 23.

Pulse-tracings in cases of aneurism showing obliquity of percussion-stroke (from left radial arteries in two cases of aneurism of the arch of the aorta).

Whilst we look upon the percussion as the *heart wave*—an indication of the shock communicated by the grasp of the ventricle upon the contained blood—we may regard the *tidal* as essentially the *blood wave*, and a measure of the volume projected through the arteries. The tidal wave is *increased* by *slow contraction of the ventricle*, by *a large volume of blood delivered through-*

* "Clinical Medicine" (London, J. and A. Churchill, 1874), p. 297.

out the arteries by the ventricular systole, and by *obstruction in the capillary circulation.* On the other hand, it is diminished by sudden contraction of the ventricle, by a small quantity of blood delivered, and by increased outflow or free circulation in the capillaries. The tracing in cases in which the tidal wave is exaggerated shows a broad, almost flat, summit; occasionally the tidal wave rises higher than the percussion up-stroke (see Figs. 24 (last tracing) and 47).*

The *dicrotic wave* is the *wave of recoil.* The onward tide of blood having more or less distended the elastic arterial channels, the sudden closing of the aortic semilunar valves imposes a barrier against any flow towards the heart; the column, therefore, rolls back towards the periphery and distends a second time the arterial channels with this dicrotic wave. The dicrotic wave is *increased* by *sudden contraction of the ventricle when the arteries are imperfectly filled*, by *increased outflow or free capillary circulation*, and by *a relaxed condition of the muscular coat of the arteries.* It is decreased by a gradual systole of the ventricle, by a state of distension of the arteries, by contraction of the arterial wall upon its contents, and by obstruction in the capillary circulation.

To estimate arterial tension.—By high tension in the arterial system is meant a condition in which the pressure of blood within the arteries is unduly great. The

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* The tidal wave should not extend beyond a straight line drawn from the summit of the percussion up-stroke to the bottom of the aortic notch. If any portion of the tidal wave encroach over such line, it is unduly developed. For many of these observations I am indebted to Dr. Mahomed's account of the Sphygmograph in Gant's "System of Surgery," 2nd ed., vol. i.

absolute amount of blood the arteries contain need not be greater, but may be much less, than normal, but the artery is tightly contracted on its contents. So the pulse may be either full, strong and incompressible, or small and wiry. One important sphygmographic evidence of increase of tension is the undue prominence of the tidal wave which we have considered, but it is a mistake to consider that this sign *alone* suffices. We

FIG. 24.

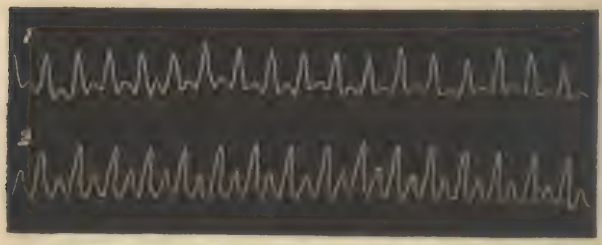
Pulses of high tension.

shall presently see that the tidal wave may be pronounced when the tension is low. The concurring signs necessary for the demonstration of high tension are—(1) in taking the sphygmogram low degrees of pressure upon the artery fail to develop the trace, whilst firmer compression succeeds; (2) the dicrotic wave has a position higher than normal in the tracing and is often but slightly pronounced (Figs. 16 and 24).

Low tension, on the other hand, means imperfect repletion of the arteries, whose muscular coat is weak

or relaxed. It is indicated by undue prominence of the dicrotic wave. We have seen that the dicrotic notch is decidedly marked in the normal pulse. In a sense, therefore, the pulse is dicrotic in health. The term "dicrotic," however, is usually applied to

FIG. 25.

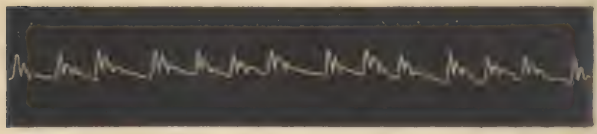


Pulses of low tension, showing full dicrotism and hyper-dicrotism. (1) from a case of pneumonia which proved fatal, (2) from a case of typhoid fever which eventually recovered.

a pulse in which the aortic notch descends abnormally low. When this notch reaches to the level of the base-line, the pulse is said to be *fully dicrotic*, and if it sink below this line it is called *hyper-dicrotic* (see Fig. 25). Such signs generally indicate serious conditions of debility.

In some tracings the line of descent is broken by many vibrations. These may be due to oscillations

FIG. 26.



Extra or supernumerary vibrations (tracing of left radial in a boy the subject of hæmophilia).

extrinsic to the artery, but in many cases they arise in the arterial wall itself. When it is very pronounced in young persons, I consider that it may be taken to indicate an abnormal condition of the arterial coats (see Fig. 26). In advanced age the condition is much more common, and, taken with other signs, indicates arterial degeneration (see some of the tracings in Fig. 24).

V.

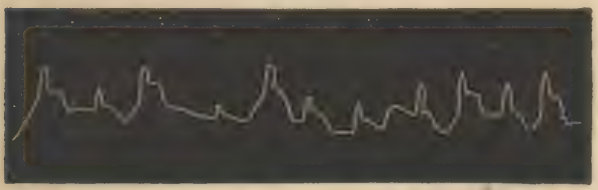
Indications afforded by the Cardiograph—Relation of cardiac revolutions to rhythm of respiration—Abortive systoles—Inversion of the trace—Interpretation of the SYSTOLIC portion of the abnormal cardiac trace—The up-stroke; its relation to the closure of the auriculo-ventricular valves—The summit and the prominence which terminates it—The down-stroke, and its relation to the closure of the semilunar valves.

INDICATIONS AFFORDED BY THE CARDIOGRAPH.—Inspection of the heart-tracing in series may show the relation of heart's action to respiration. When the breath is held the base-line is straight, but when respiration continues the curves are much more pronounced than is the case with the sphygmogram. Thus, on inspecting a heart-trace, you may find that each curve corresponds to from three to seven systoles, and these may vary much in definition and amplitude. Such variations and effacement of systoles are sometimes due to the direct intervention of the lung, in inspiration, between the heart apex and the cardiograph lever. Sometimes, this cause being excluded, the systoles are observed to be irregular or abortive. Thus, in Fig. 27 there is a systole of small volume interposed between two of larger, then follows one that is very small, and succeeding the seventh systole is an irregular rise not corresponding to a complete cycle.

A difficulty which may beset the interpretation is *inversion of the tracing*. The spot at which the heart's apex impinges in such manner as to produce bulging outwards of the chest-wall, and consequently

an ascent of the cardiograph lever, may be very limited. At the moment of systole the muscles of the ventricles draw themselves inwards towards this centre of impulse. It follows that unless the lever be exactly over this spot it may be drawn inwards

FIG. 27.



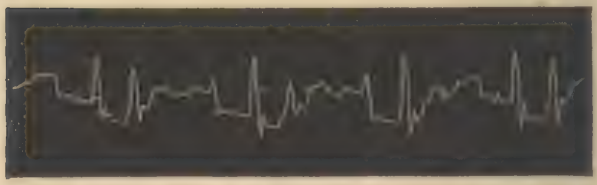
Irregular and abortive systoles. Cardiogram from a case of mitral stenosis and regurgitation, with tricuspid regurgitation.

with the motion of the ventricular wall, instead of being forced outwards at the point of maximum elongation of the ventricle; in such case the tracing becomes inverted.* A tracing of this kind may be read as a positive if it be turned upside down and examined from right to left. Or, more simply, a small mirror, such as that of an ophthalmoscope, may be placed behind it, and the reflected image read as a normal tracing. Sometimes it happens that a tracing is *partially inverted*, and then the difficulties are greater, and interpretation may be hopeless. In such case, tracings should be attempted at several situations over the impulse. From inversion of the tracing we are not justified in making any diagnosis of adherence of the pericardium as has been supposed.

* Inversion often happens when the maximum of impulse is against a rib instead of being in an intercostal space, or where the heart is covered by emphysematous lung.

Interpretation of the abnormal cardiac trace.—As with the sphygmograph trace, so with the cardiac, each element must be carefully examined. First, the *up-stroke*. This is increased in vertical height by suddenness of ventricular systole, but by no means necessarily by hypertrophy of the ventricle. In my own cases the greatest height of up-stroke was attained in a case of ulcerative endocarditis, in which the valves of the left chambers became rapidly diseased, and there were many embolisms. A reference to Fig. 28 will show how sudden and vertical the up-stroke may

FIG. 28.

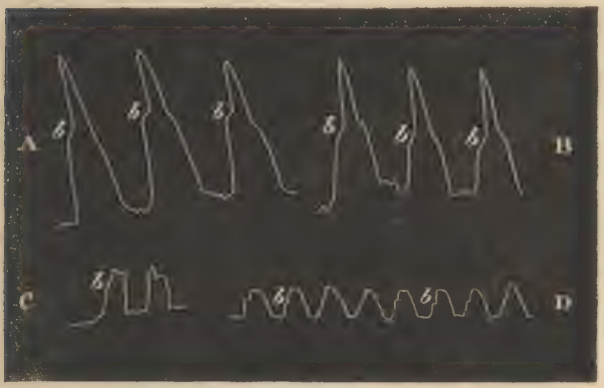


Cardiogram in a case of nervous palpitation; no organic disease.

become in conditions of palpitation. One of the loftiest up-strokes in the cardiograms figured by Dr. Galabin was in a case of exophthalmic goitre—a condition which we know to be associated with the sudden systole of a feeble ventricle; with much palpitation, in fact. In this case there was also mitral regurgitation ("Guy's Hospital Reports," 1875; Plate III. fig. 8). On the other hand, in cases of considerable hypertrophy of the left ventricle, and in aortic regurgitation, where the *pulse*-trace shows very considerable amplitude, the cardiac up-stroke may have a vertical height much less than normal.

The closure of the auriculo-ventricular valves occurs *during* the up-stroke. The first sound is heard shortly after the lever commences to ascend, and its greatest intensity appears to be at about the completion of two-thirds of the elevation. This point marks the complete closure of the auriculo-ventricular valves, and in some tracings it is rendered visible by a slight

FIG. 29.



Cardiac tracings, showing the prominence *b*, due to closure of the auriculo-ventricular valves. *A* and *B*, taken by Galabin's cardiograph, from a case of aortic obstruction and regurgitation with some mitral regurgitation. *C*, *D*, taken by Pond's instrument, *C*, from a case of mitral stenosis; *D*, from a case of mitral stenosis with regurgitation and signs of hypertrophy of the left ventricle.

interruption of the up-stroke. This is point *b* in the tracings (see Fig. 29). It does not occur when the systole is sudden. In the cardiograms figured by Dr. Galabin ("Guy's Hospital Reports," *loc. cit.*) it is seen in the hypertrophy accompanying chronic Bright's disease, in aortic disease (with regurgita-

tion), and in mitral stenosis. Of fourteen cases in which I have found it well marked, ten presented the signs of mitral stenosis, the others being instances of aortic disease.

In the next place, observe the *summit* of the trace. The extent of this summit is a measure of the *duration* of the systole. When the contraction of the ventricle is very sudden, there is an immediate fall. A reference to Fig. 28 will show that this occurs at intervals in conditions of palpitation, the tracings of intervening pulsations having broader summits. When, however, the vertical up-stroke, followed by an immediate, or almost immediate, down-stroke, is the general character of all the individual tracings, feebleness or *dilatation of the ventricle* is indicated. I have said that the undulations which are visible in the horizontal portion of the summit of the cardiac trace are probably due to the muscular movements of the ventricle. In cases of mitral regurgitation, sometimes the broad summit is formed by a vibratory line, which may be distinctly traced to the sonorous vibrations of a murmur (examples are seen in Fig. 42; see also Dr. B. Foster's "Clinical Medicine," Fig. 18, p. 509).

We have now to consider the termination of the horizontal portion of the summit in eminence *f*. In palpitation, and in cases of the sudden systole of a dilated ventricle, this eminence may be entirely lost. On the other hand, in hypertrophy of the left ventricle it is *prominent* or *rounded*.* In some cases the prominence and amplitude of eminence *f* may be found to vary considerably in successive systoles.

* I have noticed that the eminence has become markedly more rounded after the influence of digitalis.

This I have found to occur chiefly in disease of the mitral valves, most frequently in stenosis, and in the double lesion of stenosis and regurgitation; but I have also observed it in combined aortic and mitral disease. I consider prominence of *f* to indicate forcible distension of the aorta at the end of ventricular systole.

We have next to consider the downstroke, and observe whether there is any indication of the closure of the aortic semilunar valves. Practically, we may consider that the heart's second sound is synchronous with the downstroke; but in some cases the shock communicated by the reflux of blood against the valves is rendered perceptible by a notch or wave (*g*) in the tracing. This is usually near the end of the downstroke, and is here observed especially in cases in which arterial tension is low. It will be observed in the normal tracing (Fig. 13) taken when tension was low, but it is often very slightly marked. Dr. Galabin's cardiograms show it in mitral and in aortic disease. I have seen it in aortic disease, in which there is but slight regurgitation (especially where obstruction and regurgitation are combined), and in mitral stenosis. You will observe it low in the downstroke in Fig. 27, 7th, 8th, 9th, and last systoles.

VI.

Indications afforded by the Cardiograph continued—Relative duration of systole and diastole—Causes of variations—Interpretation of the DIASTOLIC portion of the abnormal cardiac trace—Difficulty of interpreting the eminence occurring immediately after the down-stroke—Its relation to suddenness of systole and suddenness of initial diastole—Line indicating rise of blood-pressure in the ventricle—Indication of auricular systole and its significance.

I HAVE said that breadth of the summit of the trace is a measure of the duration of the systole and an indication of hypertrophy of the ventricle. This indication is of more importance when it is considered relatively to the period of diastole. It is usually held that $\frac{2}{5}$ of the cardiac cycle are taken up by the ventricular systole, and $\frac{3}{5}$ by the diastole. According to the estimate of Landois, supposing the whole cycle to last 1·130 second, the systole occupies ·451 second, and the diastole ·679 second.* A measurement of the tracing of the healthy heart obtained by Galabin's cardiograph (Fig. 13) shows that whilst the systolic portion of the cycle occupies $\frac{12}{40}$ inch linear, the diastolic portion measures from $\frac{15}{40}$ to $\frac{18}{40}$. In the normal tracing obtained by Pond's instrument (Fig. 14) the systole is measured by about $\frac{6}{40}$, and the diastole by about $\frac{8}{40}$ of an inch. It is important, as evidence of a morbid condition, to note any

* Cf. Foster's "Text Book of Physiology," p. 102. London: Macmillan. 1877.

variations from the normal relations of systole and diastole observed in the cardiographic trace. The diastolic portion of the tracing may be *relatively diminished* in hypertrophy, but it is much more manifestly so in cases in which, in addition to hypertrophy, there is a condition in existence in which the ventricle becomes too rapidly filled—such occurs in aortic regurgitation and in mitral regurgitation, and, *à fortiori*, in both combined. The *diastolic portion* is relatively *increased* in the following conditions:—(*a*) When the heart's action becomes slow; "a frequent differs from an infrequent pulse chiefly by the length of the diastole."* This is exemplified by a tracing figured by Dr. Galabin,† which was taken from the exposed heart of a dog when circulation was beginning to fail—the diastolic interval is greatly prolonged (*b*) in dilatation of the ventricle (Galabin, *loc. cit.*, Plate II. Figs. 14 and 15). This may be inferred because the opposite conditions suggest, as we have seen, hypertrophy. The observation is of the highest importance as indicating in valvular lesions a want of compensation (*loc. cit.*, Plate III., Fig 5) (*c*) in mitral stenosis. This is often very markedly shown by the cardiograph. The diastolic interval, besides having characters which I shall presently describe, is greatly prolonged. Thus the condition may be indicated when it has not been rendered evident by physical signs, and the probability of combined stenosis may be shown when there are physical signs of only mitral regurgitation; so also may be determined the question of its co-existence with aortic disease (*cf.* p. 216).

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* Foster's "Text Book of Physiology," p. 109.

† "Guy's Hospital Rep." 1875, Plate I. Fig. 7.

Again, when the diastolic interval is observed to *vary greatly* in duration, as in Fig. 27, there is considerable evidence of an impeded supply to the ventricle on account of *mitral stenosis*.

Having considered the diastolic period as a whole, we will now look more closely into individual characteristics. First point *k*, the interpretation of which is undoubtedly difficult. The elevation *k* is a very marked feature in the trace, both in health and in disease. It is clearly established that it is not simultaneous with the closure of the semilunar valves, but occurs subsequently to the second sound. In the trace it is observed *after* the downstroke. If we come to consider the various morbid conditions in which eminence *k* is either increased or diminished, we find many difficulties in the way of definite conclusions. A reference to Fig. 13 might give the impression that in health it is augmented in a condition of low tension, but some of Dr. Galabin's cardiograms show that it may be also greatly augmented when arterial tension is high, as in hypertrophy of the left ventricle occurring with chronic Bright's disease. I have found it pronounced in conditions both of high and of low tension, and in disease of both mitral and aortic valves, but relatively of greater frequency in mitral lesions. On the other hand, I have found eminence *k* ill-pronounced or absent when the left ventricle has begun to fail, in the seemingly opposite condition of ventricular strength when there has been aortic disease, and again in the hypertrophy associated with Bright's disease. I think I may say that an ill-pronounced eminence *k* is considerably less common in mitral regurgitation than is the opposite condition of exaggeration, excepting in cases wherein

there are signs of failure of cardiac power or the heart's action is notably slow.

Dr. Galabin considers that eminence *k*, though not synchronous with the closure of the aortic valves, is due to a reflux of blood against the valves—an explanation to me difficult to accept, when we consider that it is by no means constantly related with increased blood-pressure in the aorta, and often is associated with an opposite condition.

Dr. Foster considers the eminence due to the first influx of blood into the ventricular cavity; and this would seem feasible, as it immediately follows the lowest point of the downstroke—*i.e.*, the maximum relaxation of the ventricle. Dr. Galabin objects that if so the elevation would not be followed by so marked a fall, an objection which does not seem to me fatal, because the entry of blood, being sudden, might produce the effect of a vibration. The frequent want of relation, however, between the magnitude of *k*, and the conditions which would increase the volume and momentum of blood entering the ventricle, are serious objections to Dr. Foster's theory. The condition which I consider to be the most constantly related with the development of eminence *k*, is *suddenness* in the action of the ventricle. Thus a reference to the cardiogram taken during palpitation (Fig. 28) will show that those systoles in which there is a high systolic rise and rapid fall, are immediately succeeded by a lofty eminence *k*, whilst the intervening cycles show a more flat diastolic period. Again, in the normal tracing (Fig. 14) the more vertical downstroke is followed by the more pronounced eminence *k*. On the other hand, the sloping downstrokes indicating a more gradual unfolding of the ventricle in Fig. 27

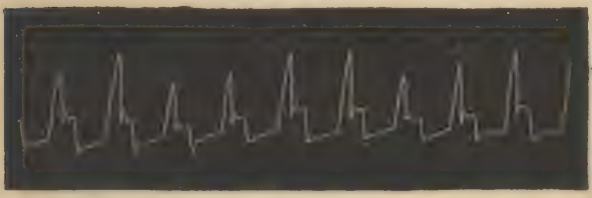
are followed by only a slight or imperceptible eminence *k*. Suddenness of action of the ventricle, and especially of diastolic relaxation, appears, therefore, to me to be the most probable cause of a high development of eminence *k*. Thus I think can be explained its magnitude in conditions of both low and high tension—in low tension when there is rapid systole and sudden relaxation of the ventricle followed by slight recoil; in high tension and ventricular hypertrophy when the diastole may be expedited by the sudden and forcible entry of blood through the coronary arteries into the heart-muscle. On the other hand, when from any cause there is slow or gradual relaxation of the ventricle, eminence *k* is ill-pronounced.

Of less difficulty is the explanation of the condition when eminence *k* is lost or merged in the elevation intervening between the downstroke and the upstroke of the following systole. This is well exemplified in Fig. 31. It indicates rapid impletion of either of the ventricles or both; it is seen in mitral regurgitation, especially when co-existing with tricuspid or with aortic regurgitation.

We turn next to line *l*, which is an indication of the filling of the cavity of the ventricle. Here, also, there is no room for doubt; the degree of ascent of the line is a measure of the degree of blood-pressure in the ventricle. If you compare Fig. 30, taken in a case of mitral regurgitation, with the healthy trace, Fig. 14, you will find that but little difference is obvious in the general characters; but the diastolic line, instead of being nearly horizontal, ascends progressively to the junction with the systolic. This, with a little broadening of the systolic portion, is the only point of

dissimilarity. These points suggest that the regurgitation causes a greater blood-pressure in the ventricle during the diastolic interval than in health, and there is compensatory hypertrophy. From this, in which the indication is but slight, turn to Fig. 31, in which you observe after eminence *k* a rapid and very marked

FIG. 30.



Cardiogram from a case presenting a soft systolic murmur at the apex, and a history of rheumatic fever three years ago, showing the line indicating the blood-pressure in the ventricle during diastole to ascend slightly. Compare with line *l*, in normal diastolic period, Fig. 14.

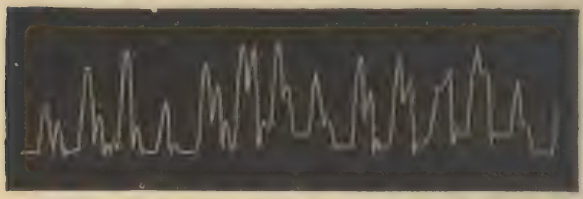
FIG. 31.

Cardiogram from a case of aortic regurgitation and obstruction combined with mitral regurgitation, showing marked ascent of the line indicating intra-ventricular pressure during diastole.

ascent—here the ventricle is filled, not gradually as in health, but by a rapid influx both from the aorta and from the left auricle, owing to the insufficiency of both aortic and mitral valves. The progressive and

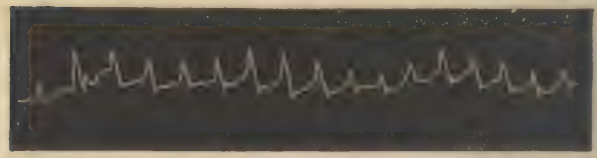
rapid rise of line *l* is chiefly seen in mitral regurgitation or aortic regurgitation, and especially when these lesions are combined; but it may be seen also in mitral stenosis, and then is an indication of considerable hypertrophy (compensatory) of the left auricle. More commonly, in mitral stenosis, the line of ascent *l* varies considerably in different cycles. This is well seen in Fig. 32, in which there is regurgita-

FIG. 32.



Cardiogram in a case presenting variable systolic and presystolic murmurs, showing variations both in systoles and diastoles.

FIG. 33.



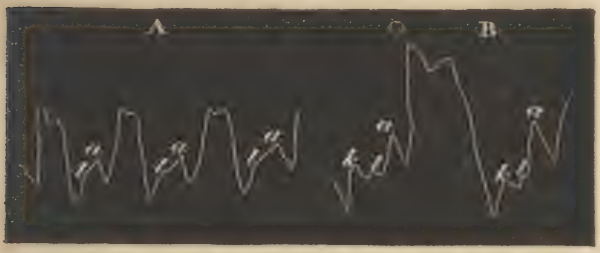
Cardiogram in a case of mitral stenosis, showing vibrations in diastolic period corresponding to presystolic murmur and thrill.

tion as well as obstruction; some of the diastolic intervals present only a sudden rise and fall, whilst others show a serrated line, which we shall consider as denoting the sonorous vibrations of a presystolic murmur, or the vibrations, sensible to the touch, of a thrill. The latter is also seen in a very marked

manner in Fig. 33. A great variation in the conditions of blood-pressure in the ventricle is also seen in Fig. 27, where tricuspid regurgitation existed as well as mitral regurgitation and stenosis.

We have next to consider the eminence *a*, which in the normal trace we have no difficulty in ascribing to the auricular systole. When this eminence is exaggerated, we have evidence of auricular hypertrophy. This is well illustrated in the two cardiograms of Fig. 34. It might be considered *à priori* probable that, inasmuch as in mitral stenosis the left auricle is usually considerably hypertrophied, the

FIG. 34.



Cardiac tracings indicating hypertrophy of the left auricle. A, taken by Dr. Mahomed from point of apex-beat by ordinary sphygmograph in a case of great hypertrophy of the left auricle with tricuspid regurgitation, but no valvular disease (*vide Med. Times and Gaz.*, April 13, 1872, Plate III. Fig. 16). B, taken by Dr. Galabin, by means of his cardiograph, in a case of mitral regurgitation, in which the left auricle was dilated and hypertrophied ("Guy's Hospital Reports," 1875, Plate III. Fig. 6).

evidence of a strong auricular systole would be a feature of the cardiographic tracings in this condition, and so would be a valuable aid to diagnosis. That in some cases of mitral stenosis exaggeration of eminence *a*, is a feature of the trace, is undoubted. This is

seen in Fig. 37 A, A, B (see also Dr. Galabin's tracings, "Guy's Hospital Reports," *loc. cit.*, Plate III. Figs. 9 and 15). A reference to the other tracings taken in cases of mitral stenosis will at once show, however, that a strong development of eminence *a* is by no means a constant feature; the reasons for this we shall presently discuss. We may premise that when in a cardiogram, from a case of mitral stenosis, eminence *a* is exaggerated and *in its normal position*, the auricle is hypertrophied, and there is no extreme narrowing of the orifice, but the momentum of blood entering the ventricle is augmented by the abnormal force of the auricular contraction.

VII.

Evidence obtained by the graphic method in valvular diseases—MITRAL STENOSIS—Sphygmographic signs—Different views as to irregularity of pulse—Interpolated pulsations—Cardiographic evidence—Explanation of interpolated pulsations—Three forms of heart-trace in mitral stenosis—Tracings showing exaggeration of auricular systole—Tracings indicating that the auricle adopts an unusual rhythm—Tracings indicating a causation of the presystolic murmur independently of the auricle.

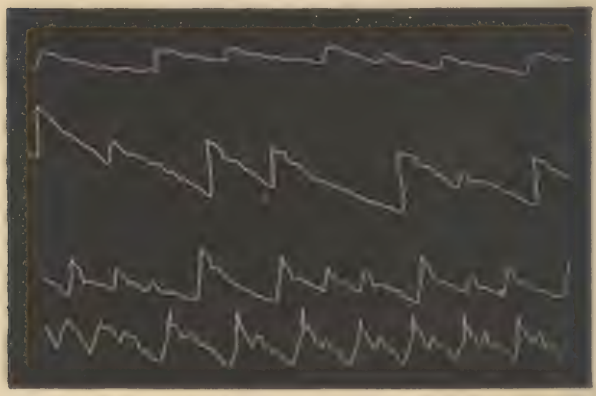
WE proceed now to consider the chief signs afforded by the sphygmograph and cardiograph in valvular diseases.

I. MITRAL STENOSIS.—The sphygmographic evidence in cases of mitral stenosis is usually very characteristic. Various opinions have been expressed as to the general characters of the pulse in this condition. For the most part observers have attributed some irregularity to the pulse, but recently Dr. Hayden has combated this view, contending that in the earlier stages of the disease "the pulse of mitral obstruction is usually quite regular, and not above ninety in the minute, but small;" and that marked irregularity occurs only in the advanced stages of the disease when the left ventricle begins to fail, and congestion of the lungs, engorgement of the right chambers of the heart, general venous obstruction, and anasarca have taken place.* This experience by no means agrees with my own, for I have generally

* "Diseases of Heart and Aorta." p. 895.

found that a notable irregularity is the distinguishing mark of the sphygmographic tracing in mitral stenosis, and that this is rendered evident in very early stages of the disease, even when there may be no sign of cardiac discomfort whatever. The first two tracings of Fig. 35 were taken from a case in which a typical presystolic murmur had become developed whilst there were no signs pointing to

FIG. 35.



The pulse-trace in three cases of mitral stenosis. The two first sphygmograms taken by Mahomed's instrument at different times in a single case; the two lower taken by Pond's instrument. All show interpolated pulsations.

cardiac mischief. It will be noted that in the topmost tracing the second pulsation is followed by a third before the base-line is reached, and in like manner the fourth is followed by the fifth. These interpolated pulsations are also seen with still more marked pronunciation in the second tracing, and here it will be noted that after the fourth pulsation there is a prolonged diastole corresponding to an inter-

mission or missed pulsation. After treatment by digitalis, the pulse was rendered slower, and the evidence of irregularity was only seen in the varying intervals between the systoles. Such peculiarity in the sphygmograms in mitral stenosis has been thoroughly recognized and described by Dr. Mahomed.* He mentions that the irregularity may be made *more* evident after the influence of digitalis. I have just asserted the converse, but I have no doubt that both propositions are true—the rhythm of the heart is altered by digitalis sometimes in the sense of developing, sometimes in that of controlling the irregularity. Muscular exercise, or any form of respiratory trouble, will develop or increase the tendency, and in any doubtful case sphygmograms should be taken in the two conditions of tranquillity and excitement after exercise.

We may conclude, therefore, that a pulse-tracing which shows irregularity in the diastolic periods sometimes missed pulsations, and, as described by Dr. B. Foster, the occasional appearance of a small abortive pulsation in the line of descent† is very strong evidence of the existence of mitral stenosis.

We may now inquire as to the probable meaning of this form of tracing. It has been considered by Dr. Foster that the rise in the line of descent is due to a premature auricular contraction which propagates itself to the ventricle; but this proposition is untenable, because each abortive pulsation is seen to consist of all the elements of a ventricular systole. It always occurs after the dicrotic wave of the tracing

* Cf. *Medical Times and Gazette*, May, 18, 1872, pp. 570 and 427, Plate IV.

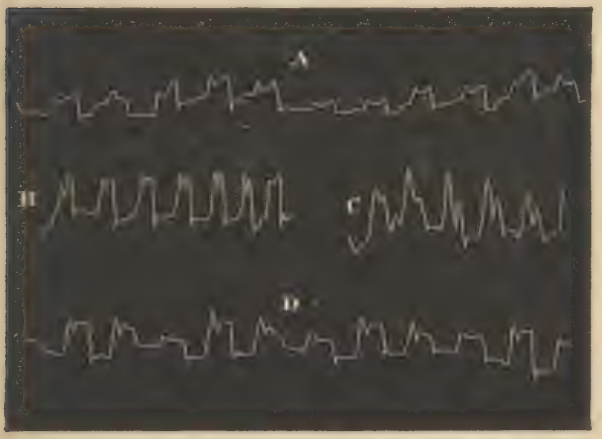
† "Clinical Medicine," p. 321.

which precedes it. It is clear, therefore, that it is an abnormal repetition of a whole cardiac cycle. The conditions of its occurrence are, according to Dr. Mahomed, these :—"The left auricle, unable to thoroughly evacuate its contents, owing to the obstruction at the mitral orifice, remains more or less full, and is generally distended at the termination of the ventricular systole, during which time it has been receiving more blood. It is thus stimulated to contract, and this it does slowly and with effort. But this is not always the case. The ventricle will not adapt itself to the altered rhythm of the auricle, but contracts immediately after the latter; and when this occurs earlier than usual, following immediately the termination of the previous contraction of the ventricle, it gives rise to another peculiarity in rhythm —namely, the secondary contraction of the ventricle." By this altered correlation between rhythm of auricle and ventricle may be explained—(1) the occasionally skipped pulsation, the long wait in diastole, and the irregularity in time of the diastolic period; (2) the supernumerary systoles. We may have slightly to modify this explanation by ascribing to tension in the passive auricle, as well as contraction of its muscle, the initiation of irregular ventricular systole.

Cardiographic Evidence in Mitral Stenosis.—Inspection of the cardiac tracing in series shows the same irregularity that is rendered evident in the tracing of the pulse. If you turn to Fig. 27, which is a cardiogram taken in the later stages of mitral stenosis, where the right heart has consecutively suffered, and there is tricuspid regurgitation, you will observe that the characteristic is notable irregularity in time and

volume. The diastolic periods greatly vary, and the systoles show differing degrees of magnitude. Turn now to the tracings in Fig. 36, where compensation is much more marked. In A and C there are varying characters of the diastolic portions, but D gives the explanation of the interpolated rise which is seen in the descending line of the sphygmogram. You will

FIG. 36.



Tracings by Pond's cardiograph in cases of mitral stenosis. A, loud, rolling, or bubbling presystolic murmur at apex. B, Typical presystolic murmur. C, Variable presystolic and systolic murmurs. D, Presystolic and systolic murmurs at apex.

notice that in the second pulsation in D, the descending line of the cardiac trace fails to reach the base-line, but is succeeded by a second pulsation, after which the line reaches its usual level; the same peculiarity is seen in the 5th and 6th pulsations. In some tracings two pulsations may be observed to

occur with no horizontal diastolic period separating them; in others only a slight eminence divides them.* It is evident, therefore, that the rise in the down-stroke of the sphygmogram is due to a complete systole of the left ventricle, the condition of the normal rhythm being so altered, that two systoles may succeed each other with a very slight or an inappreciable interval.

We will now examine the characters of the heart-tracings in mitral stenosis as *individual revolutions*. We may divide these into groups.

(1.) *Tracings in which the auricular systole is well marked.* I have already said that the only legitimate deduction from an abnormal development of the eminence *a* is *hypertrophy of the auricle*, which may or may not co-exist with mitral stenosis (see Fig. 34). Fig. 37B shows it considerably increased in a case manifesting well-marked presystolic murmur and thrill. In Fig. 36A, the auricular rise seems not only to be very pronounced, but to contribute to the general elevation normally caused by the systole of the ventricle.† In Fig. 37A it is seen that the elevation, which in all probability corresponds with the auricular contraction, has a double summit, and that this may be an indication of the vibration felt by the hand as a presystolic thrill. Similar tracings have been figured by Dr. Galabin. (Note especially Plate III. Fig. 10, in "Guy's Hospital Reports," 1875.) In one case, the period occupied by the heightened, broad-

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* *Vide* Figs. 33, 37C, where some of the systoles are divided only by a single eminence.

† This is also seen, as produced by the high tension in the auricle, in Fig. 27.

ened, and reduplicated auricular portion of the tracing could be felt by the finger to be synchronous with "a slight beat, accompanied by a thrill, just preceding the main impulse." There can be no room for doubt, therefore, that an enlarged auricular eminence may be a feature of the tracing of mitral stenosis; and further-

FIG. 37.

Tracings by Galabin's cardiograph in cases of mitral stenosis. A, A, well-marked harsh presystolic murmur at apex, with thrill. B, well-marked presystolic murmur and thrill. C, well-marked presystolic bruit, occupying greater portion of diastolic pause, at first of low pitch and "rolling" in character, becoming rasping and vibratory, and increasing in intensity up to a sudden stop with the first sound. D, presystolic murmur of low pitch (a systolic murmur also at aortic cartilage).

more, that it is an indication of the auricular-systolic causation of the murmur and thrill. A very slight consideration of the conditions would, however, suggest the probability that in many cases this auricular rise would not be manifest. The contraction of the auricle could not so influence the ventricle as to cause

it to describe an elevation if the entry of blood were impeded owing to a very marked degree of stenosis. Supposing there were no stenosis, and yet marked hypertrophy of the auricle, we might presuppose a greater rise in the auricular period than would occur in the condition of obstruction. Both these propositions are established by facts. We may conclude that if in any tracing we see a decided increase of amplitude of the auricular elevation, there is, from some cause, auricular hypertrophy; if there should be the physical signs of mitral stenosis, it is most probable that this hypertrophy is induced by the valvular lesion; if the auricular elevation is not only increased but broadened, and presenting undulations of its summit, the probabilities of mitral stenosis, even where the other physical signs are doubtful, are very considerable. We turn now to another section of cases of mitral stenosis, in which there are—

(2.) *Tracings indicating that the auricle adopts an unusual rhythm.* That the auricular systole may be modified, is proved by the tracings we have just considered; the bifid or interrupted summit of the elevation, which occupies the position of that known to be due to the auricle, leaves no room for doubt. I have observed this character in many tracings. Dr. Galabin has shown, however, that the rhythm of the auricle may be perturbed and altered in far greater degree. In the tracing (Fig. 38) it will be noticed that the two diastolic periods which are figured are abnormally lengthened. In the first of these periods are two elevations, *a*, *a*; these were found to correspond to two faint pulsations, which were not only audible but visible as slight waves in the fourth intercostal space. They were not abortive systoles, for no

sign of them was presented in the pulse-trace—there could be no doubt that they were produced by the contractions of the auricle. In the second diastolic period it is seen that the elevation indicating the auricular systole occurs, not in its normal position just before the systole of the ventricle, but nearly in the midst of the diastolic portion of the trace. It is evident, as Dr. Galabin has said, that "we have here a heart, the auricle of which sometimes contracted twice in the interval between two ventricular pulsations, and sometimes singly in the midst of a long

FIG. 38.

Heart-tracing, showing unusual rhythm adopted by the auricle. After Galabin ("Guy's Hospital Reports," 1875, p. 314, Plate II. Fig. 15).

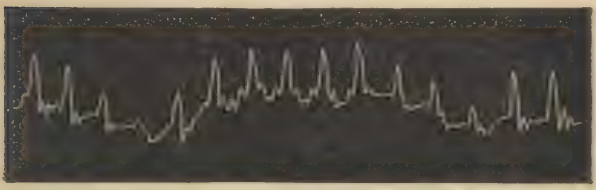
pause instead of just before the systole of the ventricle." In the case from which this cardiogram was taken, there was no proof from the other physical signs that mitral stenosis was present. Dr. Galabin has adduced it as showing that "it is *à priori* not improbable that, in mitral stenosis, the auricle, perturbed in its action by the obstruction in front, may adopt an unusual rhythm." Further experience, in my own opinion, abundantly confirms this view. If you refer to Fig. 37 c, you will observe that the line indicating the rise of blood-pressure in the ventricle is broken into several eminences and depressions, so that it

would be difficult to say which of these represented the auricular systole. In D a still stronger illustration is afforded, the diastolic period being represented by an undulatory line containing four or five eminences or swellings. In Fig. 40 you notice that the prolonged diastolic period is represented by a sinuous line more uniform and rounded. In some of the cardiograms of mitral stenosis figured by Dr. Galabin, the rise of blood-pressure in the ventricle is marked by a sudden elevation, followed by a vibratory line reaching quite up to the next ventricular systole (*loc. cit.*, Plate III. Figs. 11 and 13). The sudden rise can only be explained by the systole of the auricle, whilst the vibratory line indicates the expulsion of the blood from the auricular into the ventricular cavity. I have met with many tracings in mitral stenosis which can only be explained on the hypothesis that the auricular systole is exaggerated and occurs abnormally early. In some such tracings the line following the rise is broken only by fine vibrations; in others there are two, three, or four more pronounced elevations, such as are seen in Fig. 37 D.

In some tracings, however, in cases of mitral stenosis, it is impossible to tell where the contraction of the auricle is recorded, because the whole diastolic period is marked by a serrated or vibratory line. Good examples of this are seen in Figs. 33 and 40. It is also to be observed in Fig. 36, *b* and *d*, and in *c* in some of the diastolic intervals. It is demonstrated by many cases that these vibrations may be the graphic records of thrill or murmur, or both thrill and murmur. They are highly characteristic of the lesion; and furthermore, in my opinion, they point the lesson that the presystolic murmur is *not always* auricular-

systolic. Such view was advanced by Dr. Wilks ("Guy's Hospital Reports," 1871), who, observing that some presystolic murmurs extended throughout the whole period from the second sound to the succeeding first sound—that is, through the pause as well as through the auricular systole—considered that the first portion of such murmur was due to the blood-flow, unaided by the systole of the auricle, through the narrowed orifice. Experimental proof that such is possible has been afforded by Marey: if in an artificial schema of the circulation a perforated plug be

Fig. 39.



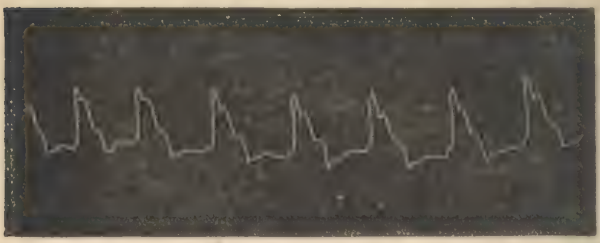
Cardiogram (by Pond's instrument) in a case of mitral stenosis, showing coarse vibrations corresponding to a presystolic murmur of low pitch and presystolic thrill.

interposed between auricle and ventricle so as to imitate the contracted mitral orifice, a *diastolic* murmur is produced when the pressure within the auricle exceeds a certain point. In such case, therefore, the murmur starts immediately from the second sound. And this seems to be only in exact accordance with probabilities —in the dilatable cavity formed by the auricle and pulmonary veins the blood, after being impelled by the right ventricle, when exit is impeded by considerable contraction of the (mitral) orifice, must be subject to pressure. The walls of the cavity into which it is forcibly injected are elastic as well as contractile, and

it is only reasonable to infer that the pent-up tension in the cavity may be a cause of the early flow into the ventricle before such flow is aided by the proper systole of the auricle. Dr. Galabin, from cardiographic evidence, has formed the opinion that the presystolic murmur may in some cases be produced independently of the auricle, and I am entirely in accord with him. The positive proof of the proposition is, I consider, afforded by the next section—viz.,

(3) *Tracings in which the auricular systole preserves its normal position.* The tracing (Fig. 40) was from a little

FIG. 40.



Cardiogram from a case of mitral stenosis with presystolic thrill and murmur, showing the auricular systole in the normal position.

girl, in whom there was a presystolic thrill with localised presystolic murmur at the apex. The child suffered from right hemichorea. In the cardiogram the auricular systole is marked in all the individual cycles, and is in its normal position just before the ventricular upstroke; it is obvious that the cause of the thrill and murmur must have been in this case independent of the auricular systole.

The cardiographic evidence, then, in mitral stenosis may be (*a*) an increase in magnitude of the elevation

denoting the auricular systole; (*b*) an increase in its breadth with a summit broken by undulations, a condition often felt by the finger as a thrill; (*c*) a perturbation of its rhythm so that it may contract at abnormal periods in the diastolic pause and may repeat its contractions; (*d*) a series of vibrations in the line denoting the rise of blood-pressure within the ventricle, often expressed also by presystolic murmur and thrill, and due sometimes to the prolonged auricular systole, sometimes to the accumulated pressure in the left auricle unaided by the contraction of the latter, and sometimes to both cases—viz., tension within the auricle at the commencement of the period of filling of the ventricle, aided subsequently by the muscular effort of the auricle.

VIII.

Graphic evidence in valvular diseases continued—MITRAL REGURGITATION—*Sphygmograms* often not characteristic—Two especial forms—(1) Pulse small, any elevation obtained with difficulty—Irregularity not observed until right chambers begin to fail—(2) Pulse ample, but of low tension—*Cardiograms* in mitral regurgitation—Ventricular hypertrophy distinguished from dilatation by breadth of summit—Vibrations caused by murmur—Occasional bifurcation of summit—Shortened diastole—Indication of auricular systole—TRICUSPID REGURGITATION—Arterial pulse-trace showing curves of respiration—Venous pulse-trace—Traces from pulsating liver—Characteristics of venous pulse-trace—The anadicrotic and katadicrotic waves—Cardiograms indicating irregularity—Right auricular systole not propagated so as to produce elevation in apex-trace, but backwards, producing anadicrotic wave in veins.

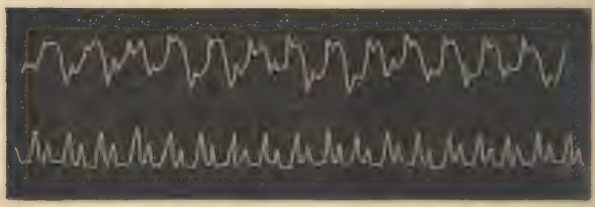
II. *Mitral Regurgitation.*—The pulse-trace in cases of mitral regurgitation is often by no means characteristic. Where compensation is fairly maintained it may present no features differing from the normal. The notable forms of the pulse altered by the condition of mitral regurgitation can, according to my observations, be divided into two classes. In one of these divisions the elevation is slight, the upstroke sloping. It is often difficult, on account of the small calibre of the artery, to obtain satisfactorily a graphic record; the systolic expansion is only represented by a slight rise, and the details of the gradually sloping downstroke are undecipherable. In such instances there is, owing to the regurgitation, only a very small

amount of blood thrown into the aorta; but the arteries are contracted upon their content. The sloping upstroke is an indication that the *point d'appui* afforded by the mitral valve in its normal state is impaired; the current which should produce the elevation flows not only towards the arteries, but backwards into the auricle. It is generally supposed that a great characteristic of the mitral pulse is *irregularity*. So far as the mere valvular lesion is concerned, I do not agree with this view. The peculiar perturbation of rhythm which I have described in mitral stenosis, I do not think occurs in uncomplicated regurgitation. On the other hand, I am perfectly aware that very marked irregularities of time and volume are oftentimes features of the pulse-traces in mitral regurgitation. These characteristics closely resemble those of the tracing, Fig. 19, which I considered to represent no valvular lesion. What, then, is their significance? I believe that they are complications of the trace induced by the secondary engorgement of the *right* chambers, by degeneration of the heart-muscle, or by both these causes combined. It is needless to say that, if this view be correct, it affords an important guide to prognosis and treatment. If in a case presenting the physical signs of mitral regurgitation you obtain a tracing showing a sloping upstroke with irregularity of time and volume, enjoin rest, and endeavour carefully to strengthen the ventricle by administering iron and digitalis; if, on the other hand, the tracing nearly approaches the normal, you may conclude that compensation is good, and anxieties at the present may be allayed. The action of digitalis, in conditions of irregularity arising from mitral regurgitation, is distinctly registered upon the

pulse-trace. It is remarkable how the disturbances of ill-compensation may be controlled, and an almost undecipherable tracing reduced from chaos to order.

The second of the forms of tracing in mitral regurgitation to which I would call attention differs greatly from the former. In this case the tracing is easily obtained, the artery not being small and contracted, and the great feature is the *pronounced dicrotic wave.* This is well marked in the pulse tracing of Fig. 41. Here the pulse is fully dicrotic, the artery being relaxed and the capillary circulation engorged. In mitral regurgitation the appearance of a strong

FIG. 41.



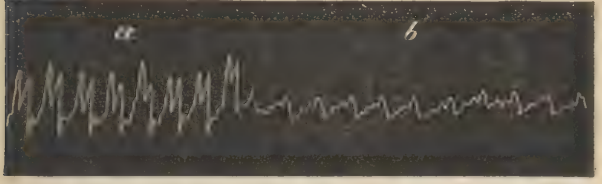
Cardiogram and sphygmogram, from a case of free mitral regurgitation.

dicrotic wave, associated sometimes with a full tidal wave, is a sign of considerable obstruction in the capillaries: such traces we meet with in cardiac dropsy.

We turn now to the *cardiac trace* in conditions of mitral regurgitation. Inspection of the systolic portion of the trace indicates whether hypertrophy predominates over dilatation. If the condition be that of dilatation of the left ventricle, the upstroke is sudden and lofty, and the descent is also rapid. If, on the other hand, hypertrophy predominates,

the *breadth* of the systolic eminence is increased. The truncated summit is sometimes broken by serrations or undulations, indications of which are seen in Figs. 30 and 36 D. I have no doubt that these may be the result of the sonorous vibrations of the systolic murmur, just as we have seen that those occupying portions of the period of diastole may correspond to the presystolic murmur. There is this difference, however, in the record of sound in the two cases—the vibrations of the presystolic murmur are only written in the exact line of the cardiac trace, whilst it would appear that the vibrations of a systolic murmur may be communicated to a much wider area of the chest-wall, so that they may inter-

FIG. 42.



Cardiogram in a case of mitral regurgitation: *a*, taken at exact apex; *b*, taken in area of loud systolic murmur, showing sonorous vibrations.

mingle, as it were, with the proper elements of the tracing. This is illustrated by Fig. 42. At *a* the trace is unmodified; but at *b*, the position of the lever being slightly altered, a number of coarse vibrations, which I consider to be due to a loud systolic murmur which was manifested in the case, are interposed. Another character which I have observed in cardiac tracings, in cases of mitral regurgitation, is the doubling or forking of the summit, which is so

marked in Fig. 42 *a*. The like is seen in Figs. 32, 36 *c*, and 41. The explanation I consider to be this: —After the first ascent of the lever, due to the hardening and rounding of the ventricle, there is a fall, because the ventricle has lost the point *d'appui* afforded by the stretched curtains of the normal valve; the continuing contraction of the ventricle, however, renews the elevation at the end of the systole.

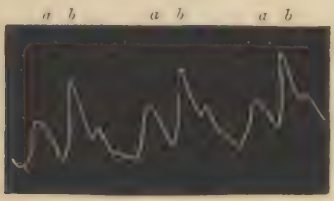
The *diastolic* portion of the trace, when there is any considerable mitral regurgitation, is shortened; the ventricle is filled more rapidly than under normal conditions. In some cases, as in Fig. 41, the rapid rise of blood-pressure causes a marked eminence, ending with a fall previously to the systole of the ventricle. In cases of mitral regurgitation, in which an increased volume of blood is impelled by a hypertrophied auricle, the eminence corresponding to the auricular systole is unduly developed (see Fig. 34).

III. *Tricuspid Regurgitation.* — The chief characteristic of the trace of the *arterial pulse* in tricuspid regurgitation is *undulation of the base-line*, marking the variations of arterial tension during respiration. If in any case manifesting much dyspnœa, and accompanied by engorgement of the right chambers of the heart, a line drawn through the bases of the upstrokes shows a series of marked curves, the probability of tricuspid regurgitation must be taken into account. So, also, in other valvular diseases, when a pronounced degree of respiratory curve is a superadded condition, it may be considered probable that the tricuspid has become insufficient.

In tricuspid regurgitation, however, graphic evidence may be obtained from the venous, as well as from the

arterial, system. A tracing may be taken of the *venous pulse* from the jugular or subclavian. Fig. 43 shows the graphic characters of such a tracing. The most conspicuous feature is the pronounced wave which precedes the main upstroke; this is due to the systole of the right auricle, which causes a reflux into the venous channels. In the descending curve is seen a sharply-defined notch, analogous to the aortic (dicrotic) notch of the arterial trace. Tracings may

FIG. 43.



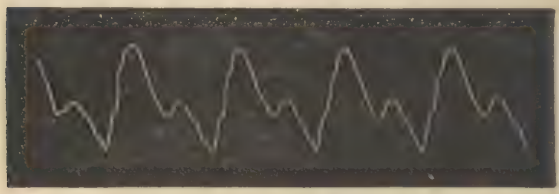
Tracing from subclavian vein in a case of tricuspid regurgitation with mitral contraction, after Galabin (*Med. Chir. Trans.*, vol. lviii. p. 365, Fig. 3); *a*, anadicrotic wave due to systole of auricle; *b*, katadicrotic wave analogous to dicrotic wave of arterial pulse.

also be taken over the seat of *pulsation of the liver* when in tricuspid regurgitation this is observed (Fig. 45). In such case the correspondence with the venous pulse will be noticed. There is the wave in the principal upstroke due to the auricular systole, and called the *anadicrotic wave*, and that in the downstroke the *katadicrotic wave*. The latter seems to be produced by like causes with those occasioning the dicrotic wave in the arterial pulse, and may be the more pronounced, as the veins are dilated and tensionless.

The CARDIAC TRACE in tricuspid regurgitation possesses no special characteristics, but in series it shows

that the systoles are irregular and unequal in volume (see Fig. 27). In the individual tracings the hypertrophy of the *right* auricle is not marked; such hypertrophy is rather indicated by the venous reflux, as shown in the tracings from the pulsating veins which we have just considered. When, therefore, you observe a very marked auricular eminence in a

FIG. 44.



Tracing from pulsating liver in tricuspid regurgitation, showing a slight anadicrotic and a pronounced katadicrotic wave.

cardiac tracing, taken in a case of tricuspid regurgitation, you should suspect hypertrophy of the *left* auricle. This is strikingly exemplified in a case recorded by Dr. Mahomed, in which there was tricuspid regurgitation, with pulsation of the large veins, in the neck, and in which the apex-tracing showed the strong auricular elevation, Fig. 34 A. The necropsy in this case showed a dilated right ventricle and wide tricuspid orifice, with greatly hypertrophied left auricle, but no valvular disease.*

* *Med. Times and Gaz.*, April 13, 1872, p. 429.

IX.

Graphic evidence in valvular diseases, concluded—AORTIC REGURGITATION—*Pulse-tracings* referred to two types—A, associated with effects of rheumatic endocarditis—High percussion upstroke—Suddenness—Effacement of dicrotic wave, and its significance—Flatness of diastolic portion.—B, associated with atheroma—Trace of high tension—*Heart-tracings*—Evidence of slowness of systole—Signs of hypertrophy of left ventricle—Shortening of diastole—Augmented blood pressure in the ventricle—Indications of diastolic thrill—AORTIC STENOSIS—*Pulse-trace* in extreme cases showing tidal wave only—Evidence when tidal wave rises above percussion—Estimation of degree of stenosis when combined with regurgitation—*Heart-trace* occasionally registers sonorous vibrations — Vibrations recorded in STENOSIS OF THE PULMONARY ARTERY.

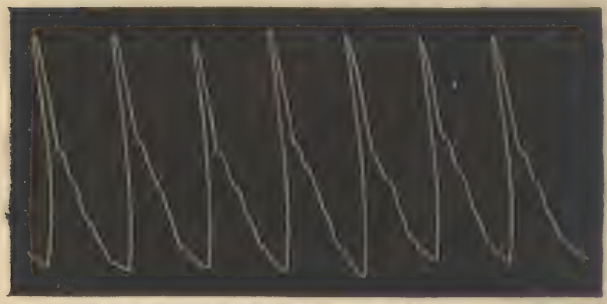
IV. AORTIC REGURGITATION. The pulse-trace which in cases of aortic regurgitation is usually highly characteristic, may be referred to one of two types, one of which (A) is usually associated with changes in the valves due to rheumatic endocarditis, and the other (B) with the incompetence due to degeneration of the aorta—aortitis deformans, or senile change.

A. The pulse-trace presents characters corresponding to the splashing, water-hammer pulse of aortic regurgitation. The first point to be noted is, that the upstroke is loftier than normal. You find that the sphygmogram in such cases is easy to take. At low pressures the tracing shows great amplitude. The grasp of the strong left ventricle impels a sudden wave into arteries, lax at the moment of systole because of

the leakage which has occurred during diastole through the imperfectly closed aortic aperture.

This *suddenness* of impulsion gives rise to the lofty percussion wave recorded by the sphygmograph (*vide* Figs. 45 and 46), as it does to the hammer-like quality of the pulse felt by the finger. Next in point of importance, as characteristic of the sphygmograms of aortic regurgitation, is *impairment or effacement of the dicrotic wave.* This would appear to be *à priori* probable. As the dicrotic wave is caused by a rebound

FIG. 45.

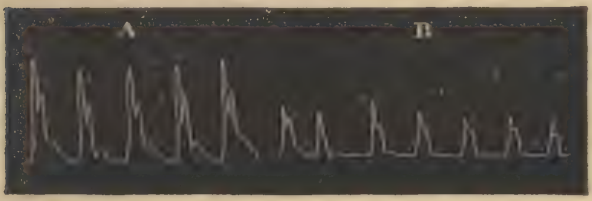


Tracing from Brachial Artery in a case of free aortic regurgitation, showing effacement of dicrotic wave.

from the closed extremity of the aorta, it is only reasonable to infer that, if the closure be imperfect, the dicrotic wave will also be ill-pronounced. That it may be so is proved by many tracings (*vide* Fig. 45). In the early days of the application of the sphygmograph it was considered that this impairment of the dicrotic wave was the chief sign of aortic regurgitation; such view is not, however, borne out by facts. As a positive sign, effacement of dicrotism is of very high value, for it indicates very free aortic regurgita-

tion. It must not be concluded, however, that the presence of a marked dicrotic wave *negatives* regurgitation. In Fig. 46 A, you notice that the tracing of the radial, taken with Pond's instrument, has great altitude of percussion, the characteristic of aortic regurgitation: there is a sharply defined tidal wave, and near the base of the tracing is a well-marked dicrotic wave. In this case there was undoubted aortic regurgitation, and it is well proved that in such cases a pronounced dicrotic wave may be recorded, the partially-closed aortic cusps forming a sufficient *point*

FIG. 46.



Tracing from radial artery in aortic regurgitation: A, taken with light pressure showing dicrotic wave near the base-line; B, with slightly increased pressure showing obliteration of dicrotic wave.

d'appui for its production. It will be noticed, however, that in the tracings in which the percussion upstroke is high, the dicrotic wave, though it may be amply developed, is low or near the base-line. Another point of importance is its easy obliteration by pressure. In the type of aortic regurgitation, which we are now considering, I have said the elements of the trace are best brought out by low pressure, then the dicrotic wave may be full and rounded; but if the pressure upon the artery be only slightly increased, the wave may be altogether obliterated. This characteristic of

the pulse of aortic regurgitation is demonstrated with great facility by Pond's sphygmograph. You commence to take the trace with low pressure, and all the elements of the trace become well marked, as seen in Fig. 46 A, which shows the dicrotic wave full and rounded. Then if, whilst the slide is travelling, you slightly increase your pressure upon the artery, you find that the dicrotism is obliterated, as shown in Fig. 46 B, the diastolic portion of the tracing being rendered flat. We may conclude, then, as regards those sphygmograms of aortic regurgitation which present a high elevation that the dicrotic wave is either obviously impaired or else, though apparently pronounced, is *easily obliterated by increase of pressure*. This leads us to the third characteristic of such pulse traces, which is *flatness of the diastolic portion*. The line succeeding the dicrotic notch is low in the tracing or is horizontal. Such flatness indicates the emptiness of the artery during ventricular diastole.

The second type, B, of pulse-trace in aortic regurgitation differs considerably from the preceding: there is no notable exaggeration of the percussion upstroke with rapid fall, but a resemblance with the tracings indicating *high tension* in the arteries, as in Fig. 24. In such case the summit of the trace is broadened, the tidal wave being sustained; the dicrotic notch, though ill-pronounced, is high in the tracing; and the gradually-sloping line corresponding to the diastolic period, tells of peripheral obstruction. As the former type of pulse was chiefly associated with the changes in the aortic valves induced by rheumatic endocarditis, so this is seen for the most part in conjunction with *degenerative diseases* of the aorta and arteries.

So the sphygmograph becomes a valuable means of

diagnosis as regards the two pathological conditions. For example: a case presents itself with the physical signs of aortic regurgitation, the patient being past the prime of life, and giving a negative or obscure history of rheumatism. Your sphygmographic tracing is not of the typical form Fig. 47 A, and you are only able to conclude from it that there is *high tension in the arteries*. Such conclusion is of the highest importance. It either negatives the conclusion that the changes are due to rheumatic endocarditis, or indicates that any regurgitation so produced is slight and well compensated. But further, in a majority of cases it goes to prove that the condition giving rise to the aortic regurgitation is due to atheroma of the aorta—to endarteritis or aortitis deformans.*

The CARDIAC TRACE in aortic regurgitation does not present such important characters in regard to diagnosis as the pulse trace, but nevertheless affords evidence of much value. In the first place, it is noticeable that the quality of *suddenness*, so marked in the majority of the sphygmograms, is not a feature of the cardiograms. In a case in which the sphygmograph indicates a lofty, abrupt percussion wave, the cardiograph demonstrates a slow and prolonged ventricular systole. Dr. Galabin has proved by accurate measurement, that in a pulse-curve taken in a case of aortic regurgitation, in which the general shape differed little from the normal, the upstroke occupied about one-third less time than that of the healthy pulse.† On the other hand, "the interval occupied

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* *Cf.* Mahomed, *Med. Times and Gaz.*, Sep. 21, 1872, p. 325, and "Guy's Hospital Reports," 1879, p. 419.

† Galabin, *Med. Chir. Trans.*, vol. lix. p. 361.

by the main upstroke of the trace, which indicates the time which the ventricles take in becoming fully hardened, may be increased in cases of aortic disease from its normal value of about one-twelfth of a second to as much as from one-eighth to one-sixth of a second; that is to say, that it may be nearly doubled."* The systolic portion of the tracing is usually broad, and the eminence *f* well-marked and rounded—indications of hypertrophy of the left ventricle. In some cases, however, the opposite condition prevails, the summit is not truncated but narrow, and the diastolic fall rapid; this is an indication that *dilatation* of the left ventricle prevails. As regards the diastolic portion of the tracing, it is very important to note its *relative duration*. It is common to find in aortic regurgitation that this is much shortened. There may be scarcely any interval between one systolic upstroke and the next, or only a slight single eminence; this is an indication that the regurgitation is very free, for the ventricle becomes rapidly filled. Or the diastolic portion, instead of being horizontal, may present an ascending line (as in Fig. 31), indicating that the blood-pressure in the ventricle during diastole becomes progressively increased; this is notably the case where mitral regurgitation, especially when combined with hypertrophy of the left auricle, co-exists. Occasionally, vibrations corresponding to diastolic thrill are marked upon the trace. Such is shown in certain of the cardiograms recorded by Dr. Galabin ("Guy's Hospital Reports," 1875, Plate II. Figs. 9 and 10); and I have observed it in many others.

In Aortic Obstruction, when the lesion is

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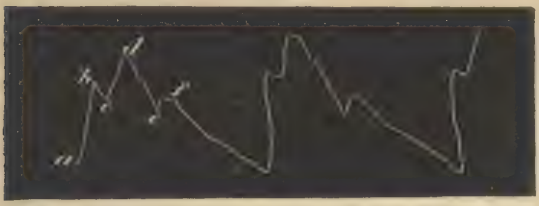
* *Loc. cit.*, p. 363.

extreme, the *pulse trace* may possess highly distinctive characters. The percussion wave is effaced, for the artery, owing to the narrowing of the aorta, is slowly and gradually distended by the ventricular systole. The tracing in such cases shows sloping upstrokes and downstrokes resembling that obtained sometimes in cases of aneurism (see Fig. 22). Such tracings are figured by Dr. Mahomed (*Medical Times and Gazette*, Aug. 10, 1872, Plate V.) and by Dr. Hayden ("Diseases of Heart and Aorta," p. 869, Fig. LII.). This last was in the case of a boy aged seven and a half years. In these cases there is only one element of the trace, the tidal wave, all else being obliterated. In other cases the percussion upstroke

Fig. 47.



Sphygmogram in case of aortic obstruction (Dr. Galabin). A large tidal wave, *c d*, rises high above the percussion wave, *a b*, indicating prolonged ventricular contraction; there is a marked dicrotic wave, *e f*, indicating that there is little or no reflux through the aortic orifice; pulse very slow.

is arrested, and from its point of arrest proceeds an ample tidal wave, which rises high above the percussion wave.* This is seen in Fig. 47. There is not such extreme narrowing of the aortic orifice that the element of suddenness is lost, but the percussion

* *Cf.* Galabin, *Med. Chir. Trans.*, vol. lix. p. 384.

is short, and the pronounced tidal wave is an indication of the prolonged systole of the ventricle. So important do I consider this form of tracing, that I would say, whenever you meet with a radial tracing in which the percussion wave is suddenly arrested and an ample tidal wave rises above it, suspect aortic stenosis.* I have before said that the signs of this lesion are often obscure, and that during life it often remains undiscovered. As an aid to the diagnosis of the condition, I think the sphygmograph is of great value. So also in combined aortic regurgitation and obstruction, a conjunction which is much more common than either affection singly, you may take the amplitude of the tidal wave as a measure of the degree of stenosis. A pulse trace, which at very light pressure has the typical verticality of aortic regurgitation with very slight pronunciation of the tidal wave, may, by increase of pressure, be made more and more to develop the tidal, and this I consider to be an indication of the accompanying stenosis. In certain cases of aortic stenosis there is a marked division between the percussion and tidal waves, so that the summit of the trace is forked (*vide* Fig. 47). Such tracings are figured by Dr. Mahomed (*Medical Times and Gazette*, Aug. 10, 1872; Plate V. Figs. 47, 48, and 49); they may explain the thrill which is sometimes felt in the pulse of aortic stenosis.

Cardiographic Tracings in aortic stenosis are not often characteristic. Traces taken by Pond's instrument with very light pressure, over the site of an aortic systolic murmur, may present vibrations caused by the conditions which produce the sound.

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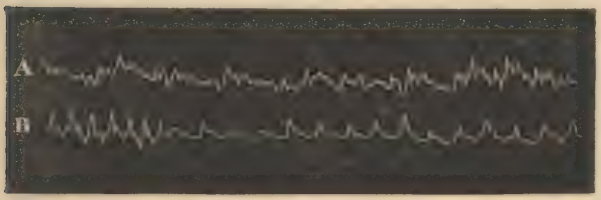
* *Vide* Fig. 24, the last tracing.

This is strongly insisted upon by Dr. Pond as a point of value, but considerable caution must be used in so accepting it. These fine vibrations may have many causes—tremulous movements of the skin, vibratory contractions of the intercostal muscles, and rhonchi, for example. Nevertheless, I have had experience that such vibrations may be recorded in the area of a systolic aortic murmur, just as I have already shown them to be made manifest in mitral regurgitation (see Fig. 42) and in mitral stenosis (Figs. 33, 36, 37, and 39).

In STENOSIS OF THE PULMONARY ARTERY also

FIG. 48.



Cardiogram from a case of congenital Cyanosis with murmur over the site of the pulmonary artery: A, showing vibrations over the maximum of a loud superficial systolic murmur; B, tracing at the apex of the heart.

such vibrations, due to a systolic murmur, may be registered upon the trace, as in the example Fig. 48.

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NOTE.—The figures in darker type (*e.g.*, **123**) refer to Part II.

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